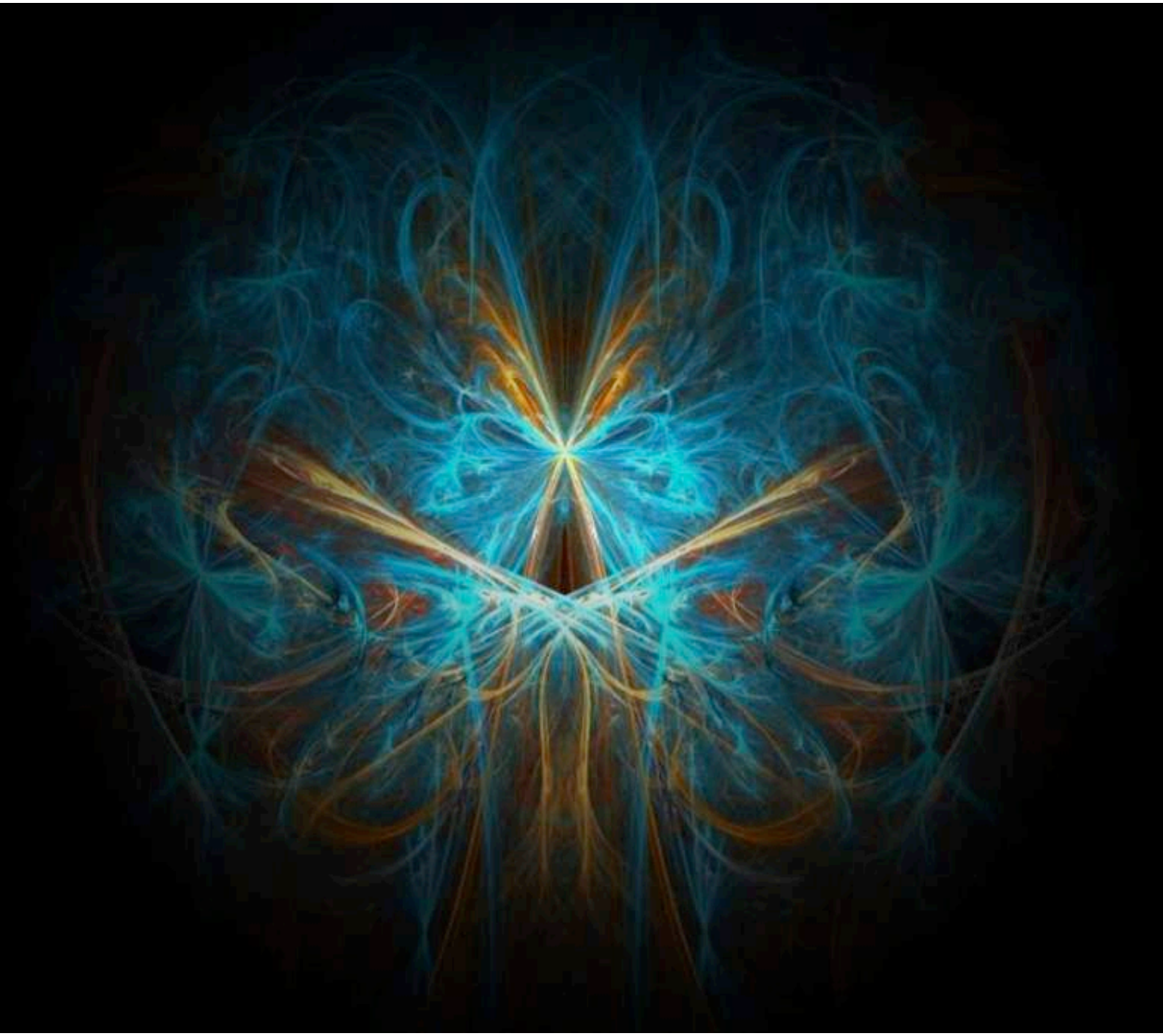
AUSTRALASIAN CHRONOBIOLOGY SOCIETY

12th Annual Scientific Meeting 20th October 2015



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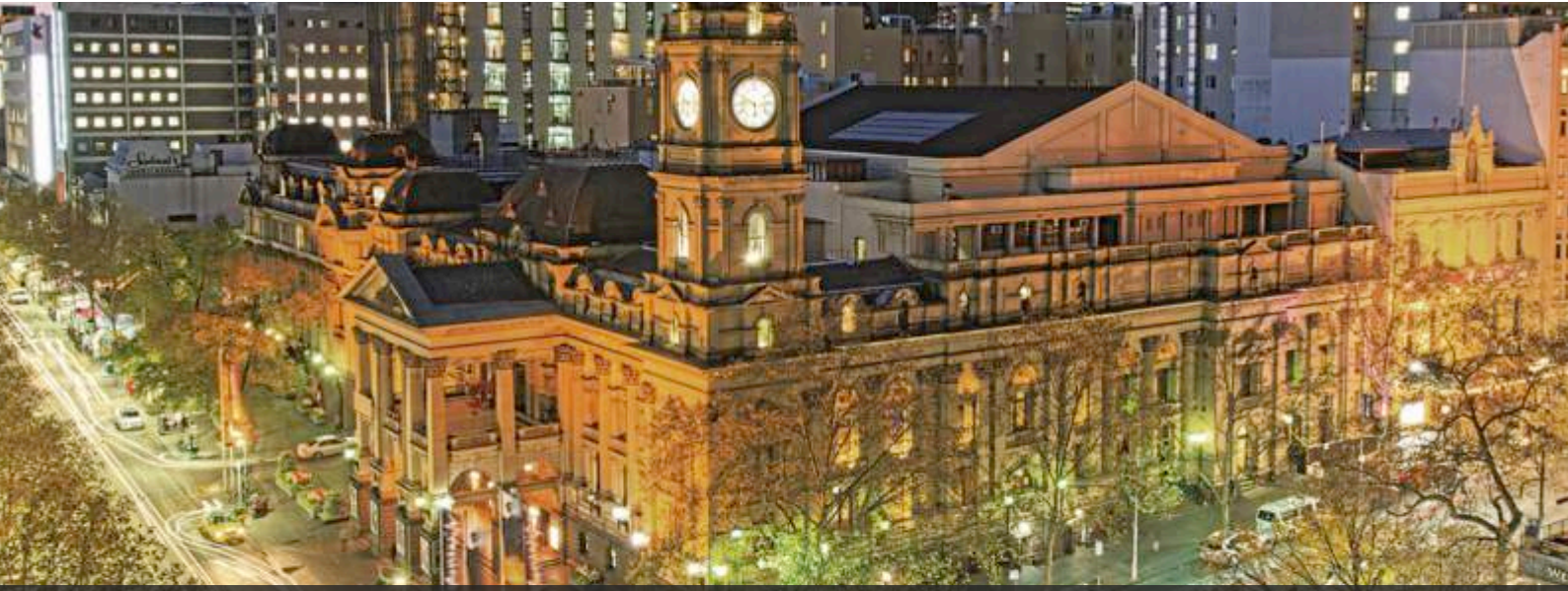

The 12th Annual Scientific Meeting

The Australasian Chronobiology Society was founded in early 2004. Our annual scientific meeting brings together Australian and international researchers interested in the influence of 24-hour circadian rhythms on behaviour, cognition, sleep and health. The 12th Annual Scientific Meeting in 2015 is being held in Melbourne, at the Melbourne City Town Hall.

Pictured: The Melbourne City Town Hall

Keynote Speaker

Professor Kenneth P. Wright Jr.

Professor Wright joins us from the Department of Integrative Physiology at the University of Colorado in the United States. There, he is the Director of the Sleep and Chronobiology laboratory.

His research interests include health and safety consequences of insufficient sleep and circadian misalignment and the development of countermeasures and treatments for sleep and circadian disruption to improve public health and safety.

He will discuss the effects of caffeine on circadian timing, a topic he recently published on in *Science Translational Medicine*.

PROGRAM

9:00 am	**Arrival tea and coffee**	
9:30 am	Sean Cain	Welcome
9:40 am	Kenneth Wright *University of Colorado (US)*	Caffeine and Circadian Timing
10:10 am	Steve Lockley *Harvard University (US)*	Tasimelteon for the treatment of non-24-hour sleep-wake rhythm disorder in the totally blind
10:45 am	**Morning Tea**	
11:15 am	Leon Lack *Flinders University (SA)*	Flipping the 'Sleep Switch' activates Process 'O' (Onset/Offset) to produce alerting benefits of brief naps as well as more sustained sleep after nocturnal awakenings
11:35 am	Stephen Pittman *Phillips (US)*	Innovation, translational research and circadian diagnostics in clinical practice
11:55 am	Shantha Rajaratnam *Monash University (VIC)*	Melatonin in human circadian rhythms
12:15 pm	**Lunch**	
1:00 pm	Sean Cain *Monash University (VIC)*	Using the pupillary light reflex to distinguish circadian and non-circadian DSPD types
1:10 pm	Tracey Sletten *Monash University (VIC)*	The role of circadian rhythm phase in individual responses to sleep restriction
1:20 pm	Greg Willis *Bronowski Institute (VIC)*	Further evidence for the existence of a functional link between the retina, the nigra in movement and movement disorders.
1:30 pm	Oliver Rawashdeh *University of Queensland (QLD)*	The circadian clock protein PERIOD1 regulates memory-relevant signaling in the mouse hippocampus
1:40 pm	Siobhan Banks *University of South Australia (SA)*	Impacts of eating at night on driving performance
1:50 pm	Raewyn Poulsen *University of Auckland (NZ)*	The chondrocyte-intrinsic circadian clock is disrupted in osteoarthritis
2:00 pm	Rachel Schembri *RMIT/Austin Health (VIC)*	The effect of sleep apnea severity on neuropsychological function in people with acute quadriplegia and obstructive sleep apnea
2:10 pm	Nicole Lovato *Flinders University (SA)*	Estimating circadian phase in patients with Delayed Sleep Phase Disorder
2:20 pm	Claire Ellender *Melb. Sleep Disorders Centre (VIC)*	Long-term sleep measurement: unlocking the rhythms of life
2:40 pm	**Afternoon Tea**	
	Data Blitz Presentations	
3:00 pm	Joanne Carpenter *University of Sydney (NSW)*	Sleep-wake profiles, and associations with cognitive functioning in young people with affective disorders
3:05 pm	Emily Zou *RMIT (VIC)*	Sleep-dependent recall and recognition of emotional visual images
3:10 pm	Zarraar Zia *Monash University (VIC)*	Sleep in pregnancy and its association with birth weight: a systematic review
3:15 pm	Joshua Miles *University of Melbourne (VIC)*	Depression in Obstructive Sleep Apnea and the role of emotional regulation
3:20 pm	Jade Murray *Monash University (VIC)*	Characteristics of sleep in Delayed Sleep Phase Disorder
3:25 pm	Gorica Micic *Flinders University (VIC)*	Circadian tau differences in biological, behavioural and sleepiness rhythms in Delayed Sleep-Wake Phase Disorder and Non-24-hour Sleep-Wake Disorder patients
3:30 pm	Anastasi Kosmadopoulos *The Appleton Institute (SA)*	Deriving non-wear time thresholds for sleep/wake accelerometers based on patterns of bedtime quiescence
3:35 pm	Thomas Kontou *The Appleton Institute (SA)*	Glucose metabolism is impaired after one week of sleep restricted circadian misalignment
3:40 pm	Yaroslava King *The Appleton Institute (SA)*	What is the relationship between social support, child sleep and postnatal depression?
3:45 pm	Raymond Matthews *The Appleton Institute (SA)*	Too Hot, Too Tired: The Effects of Temperature and Sleep Restriction on Firefighters' Simulated Driving Performance
3:50 pm	Leilah Grant *Monash University (VIC)*	Stroop performance after one night of sleep deprivation: effect of BDNF genotype
3:55 pm	Crystal Grant *University of South Australia (SA)*	During 50hrs of sleep deprivation caffeine decreases perceived sleepiness, however, does caffeine consumption alter hunger and satiety ratings?
4:00 pm	Madeline Stainsby *RMIT (VIC)*	Reducing sleep-onset latency in pre-school children using slow-frequency tones: a pilot study
4:05 pm	Maggie Yu *RMIT (VIC)*	A pilot study investigating the effectiveness of listening to a metronome sound that can help children with autism initiate sleep
4:10 pm	Thuy Dang *RMIT (VIC)*	Panax ginseng as an adjunct to the continuous positive airway pressure therapy in the management of obstructive sleep apnoea
4:15 pm	Elise McGlashan *Monash University (VIC)*	Antidepressant medication increases the response of the circadian system to light

SPONSORSHIP

Gold Level

The Monash Institute for Cognitive and Clinical Neurosciences (MICCN) brings together world-class researchers with cutting-edge research infrastructure from a range of disciplines and faculties. The result – a unique institute capable of revolutionary insight and advancement in areas of critical importance to well-being including: Addiction; Attention and Memory; and Sleep. MICCN openly and actively shares insights and advances with other researchers, the neuroscience community, industry and beyond.

MICCN is embedded with Monash's world leading Faculty of Medicine, Nursing and Health Sciences. It is the largest institute of it's kind in Australia and the largest grouping of cognitive and clinical neuroscientists in the Asia-Pacific region.

As agents of change, MICCN works with our staff, our clients, our research participants and involves the wider public in the discovery process. This ensures that MICCN research is linked to societal needs and that findings inform debate, so that changes can enhance our lives and enrich our culture.
Our research and education programs are delivered in collaboration with clinical and industry partners, ensuring the next generation of cognitive and clinical neuroscientists are as skilled in the clinic as they are at the bench, as useful to business as they are to academia. These are exciting times for neuroscience and for MICCN.

We hope you will join us on our journey towards understanding how *the human brain is behind everything we do*.

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Monash Institute of Cognitive and Clinical Neurosciences

Silver Level

Servier is a privately owned pharmaceutical company with a long-standing commitment to research and development. In 2005, company founder Dr Jacques Servier announced that all profit from Servier worldwide operations is now channelled into research and development projects through the Servier Foundation. Servier Australia's commercial interests are presently in cardiovascular disease (Coveram - perindopril and amlodipine, Coversyl - perindopril arginine, Coversyl Plus – perindopril arginine/indapamide, and Coralan - ivabradine) and more recently, major depression with the registration of Valdoxan (agomelatine).

SERVIER

Silver Level

At Philips, we look beyond technology to the **experiences of patients, physicians and caregivers** across the health continuum from healthy living to prevention, **diagnosis, treatment and recovery**. We unlock insights leading to meaningful innovations from hospital to home. Our solutions combine clinical breadth and depth of expertise, technology and services, actionable data, consultative new business models and partnerships. Together, with our customers, we take risks and share responsibility – so that we can **transform how care is delivered** and experienced. It's a unique perspective empowering us all to create a **healthier future.**

Bronze Level

The BMedical Group comprises of BMedical Pty Ltd and Homemed.com.au. BMedical supplies innovative diagnostic and treatment products for sleep disorders, fatigue research And industrial health throughout Australia and New Zealand. BMedical's sleep related product range includes: **Provent Sleep Apnea Therapy, Philips Actiwatches, B-Alert Wireless EEG, Apnea Guard, ARES Home Sleep Testing, SleepProfiler for diagnosing insomnia and ingestible core body temperature monitoring.** Homemed.com.au is a new web-based store for the BMedical group offering a complete selection of non prescription sleep treatment and health monitoring products. These include **Theravent Snore Therapy, SHUTi online CBTi (insomnia treatment)** and **Feel Bright Light** for circadian disorders, shiftwork sleep adjustment and jetlag. To shop or learn more visit **www.homemed.com.au**

The Alertness CRC is an industry focused research program committed to maximising alertness in the workplace. The mission of the Alertness CRC is to:

1. Promote the prevention and control of sleep loss and sleep disorders
2. Develop new tools and products for individuals and organisations to improve alertness, productivity and safety.

A Cooperative Research Centre (CRC) is the ideal mechanism to bring together stakeholders in a multi-sector, end user driven initiative to address key challenges.

DATA BLITZ ABSTRACTS

Sleep-wake/circadian profiles, and associations with cognitive functioning in young people with affective disorders.

Joanne S. Carpenter[1], Rébecca Robillard[1], Rico S. C. Lee[1], Daniel F. Hermens[1], Sharon L. Naismith[1], Django White[1], Bradley Whitwell[1], Elizabeth M. Scott[1] and Ian B. Hickie[1]

[1] Clinical Research Unit, Brain and Mind Centre, University of Sydney, Camperdown, NSW

Background: Early-stage affective disorders are associated with sleep-wake/circadian rhythm disturbances, however these disturbances are considerably heterogeneous. Cognitive dysfunction is also associated with affective disorders but interactions with sleep-wake disruptions are not well understood.
Aims: To characterise profiles of sleep/circadian disturbance in young people with affective disorders and examine associations between these profiles and cognition.
Methods: Actigraphy monitoring was completed in 152 young people with affective disorders, and 69 healthy controls. Patients also underwent detailed neuropsychological assessment. Actigraphy data were processed to estimate both sleep and circadian parameters.
Results: Two hierarchical cluster analyses identified distinct patient groups. Three clusters based on sleep variables included a 'long sleep', a 'disrupted sleep', and a 'delayed and disrupted sleep' cluster. Three clusters based on circadian variables included a 'strong circadian', a 'weak circadian', and a 'delayed circadian' cluster. Medication use differed between clusters. The 'long sleep' cluster displayed significantly worse visual memory performance compared to the 'disrupted sleep' cluster. No other cognitive functions differed between clusters.
Conclusions: These results highlight the heterogeneity of sleep/circadian profiles in young people with affective disorders, and provide preliminary evidence in support of a relationship between sleep and visual memory, which may be mediated by use of antipsychotic medication.

Sleep-dependent recall and recognition of emotional visual images.

Emily Zou[1] and Russell Conduit[1]

[1]School of Health Sciences, RMIT University, Melbourne, VIC

Evidence from behavioural and neurophysiological studies demonstrate that sleep, relative to the corresponding period of wakefulness, benefits memory consolidation and subsequent retrieval. Independently, memory is also modulated by emotion, leading to enhanced consolidation of emotionally salient stimuli compared to neutral stimuli. Studies that have investigated the effects of sleep on emotional memory consolidation report mixed findings, suggesting that the effects of sleep on memory could be subtle and fragile, and may only emerge under specific experimental conditions. This study aims to investigate the effect of midday naps on emotional memory consolidation by using two different modes of retrieval – free-recall and recognition – to potentially shed light on task-factors which determine sleep-dependent learning. We replicated Nishida, Pearsall, Buckner, and Walker (2009) nap paradigm. Participants completed two learning phases (at 1:00pm, and 5:00pm) and a recognition test (at 5:15pm). Participants in the nap group had a 90-minute nap opportunity after the first learning phase and recall was tested 30-minutes after nap to eliminate the effects of sleep inertia. Similar, protocol was followed for the no-nap control group with the exception that recall occurred at 3:00pm. The results of this study potentially have implications in the way future sleep-dependent learning research is conducted.

Sleep in pregnancy and its association with birth weight: a systematic review

Zarraar Zia[1,2], Michelle L Blumfield[2], Sean W. Cain[3], Helen Truby[2]

[1] School of Medicine, Faculty of Medicine, Nursing and Health Sciences, Monash University, Australia
[2] Department of Nutrition and Dietetics, Faculty of Medicine, Nursing and Health Sciences, Monash University, Australia
[3] School of Psychological Sciences, Faculty of Medicine, Nursing and Health Sciences, Monash University, Australia

Reduced sleep is an independent risk factor for obesity in infants, children and adults. This study summarizes the associations between sleep deprivation during pregnancy and infant birthweight. A systematic review of articles in MEDLINE, CINAHL, Cochrane Library and PsychINFO, without date limits. Studies that reported sleep variables in a pregnant population and subsequent birthweight of infant were included. Two authors independently assessed the methodological quality of each included study using the Quality Assessment Tool for Quantitative Studies[1]. Data was extracted from 10 cohort studies; of which 4 (involving a total 2925 women) described the association of maternal sleep disruption on birth weight with statistical significance. There were contradictory results; 2 high quality studies showed sleep disruption was associated with decreased birthweight and 2 studies, of lower quality, reporting an increased birthweight. These discrepancies may be attributed to poor study design (lack of validated sleep measurement tools), anthropometric differences within different ethnicities and cultural norms regarding sleep hygiene within each population. Sleep deprivation is associated with a change in birthweight; however these results are not necessarily generalizable to the Australian population. Further exploration of the relationship between maternal sleep and birth weight is required to inform appropriate advice.

Depression in Obstructive Sleep Apnea and the Role of Emotional Regulation

Joshua C. Miles[1,2] Melinda L. Jackson[2,3] Christian L. Nicholas[1] V Vien Lee[2,3] Maree Barnes[2] Julie Tolson[1,2]

[1]Melbourne School of Psychological Sciences, University of Melbourne, Melbourne, Australia
[2]Institute for Breathing and Sleep, Austin Health, Melbourne, Australia
[3]School of Health Sciences, RMIT University, Melbourne, Australia

Obstructive Sleep Apnea (OSA) is associated with high rates of depression, but the relationship between these two conditions is unclear. Previous studies have identified sleepiness and fatigue as possible contributors, with disease severity having little relationship to depression symptoms. The aim of this study was to examine whether emotional regulation mediates the relationship between OSA and depressive symptoms. Forty-eight OSA participants (18 Female, M_{age}=46.5, SD_{age}=11.8) and 21 healthy controls (6 Female, M_{age}=32.7, SD_{age}=6.5) completed a diagnostic polysomnogram, the Centre for Epidemiological Studies Depression Scale (CES-D), Epworth Sleepiness Scale (ESS) and Difficulties with Emotional Regulation Scale (DERS). OSA participants demonstrated significantly higher CES-D ($p<0.001$) and DERS ($p<0.05$) scores compared to controls. Of the DERS factors, only Lack of Emotional Awareness demonstrated a significant difference between OSA and controls ($p<0.01$). Mediation by DERS did not significantly predict CES-D with the Apnea/Hypopnea Index, Body Mass Index and ESS scores. People with OSA have difficulty regulating their emotions driven by a lack of emotional awareness. Despite a lack of relationship between DERS and OSA measures, DERS did predict CES-D scores indicating that improving emotional awareness may reduce depressive symptoms in OSA patients.

DATA BLITZ ABSTRACTS

Characteristics of sleep in Delayed Sleep Phase Disorder

Jade M. Murray[1,2,3], Tracey L. Sletten[1,2,3], Michelle Magee[1,2,3], Christopher Gordon[2,3,4,5], Nicole Lovato[2,6], Delwyn J. Bartlett[2,3,4], David J. Kennaway[7], Leon C. Lack[2,6], Ronald R. Grunstein[2,3,4], Steven W. Lockley[1,2,3,8,9], & Shantha M.W. Rajaratnam[1,2,3,8,9]

[1]School of Psychological Sciences, Monash University, Clayton, Victoria, Australia
[2]Cooperative Research Centre for Alertness, Safety and Productivity, Clayton, Victoria, Australia
[3]NHMRC Centre for Sleep and Circadian Neurobiology, Sydney, New South Wales, Australia
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[6]Adelaide Institute for Sleep Health, School of Medicine, Flinders University, Adelaide, South Australia
[7]Robinson Research Institute, School of Pediatrics and Reproductive Health, University of Adelaide, Adelaide, South Australia, Australia
[8]Division of Sleep and Circadian Disorders, Departments of Medicine and Neurology, Brigham and Women's Hospital; Division of Sleep Medicine, Harvard Medical School, Boston, MA, USA
[9]Division of Sleep Medicine, Department of Medicine, Harvard Medical School, Boston, Massachusetts, USA

Whilst the underlying mechanism of Delayed Sleep Phase Disorder (DSPD) is a delay in the timing of the endogenous circadian pacemaker, circadian phase measurement is not a requirement of diagnosis and therefore many individuals without a delay in endogenous circadian rhythms are misclassified as DSPD. Current diagnostic criteria rely exclusively on self-report sleep-wake information, making it difficult to determine the presence and extent of circadian phase misalignment, often leading to suboptimal management or mistimed treatment. This study aimed to compare subjective sleep-wake characteristics in DSPD patients with and without circadian phase delay relative to desired bedtime (i.e., circadian *vs.* non-circadian DSPD). Circadian DSPD was defined as salivary dim light melatonin onset (DLMO) occurring at or after desired bedtime and non-circadian DSPD was defined as DLMO occurring before desired bedtime. A total of 185 participants who met diagnostic criteria for DSPD undertook 7 days of sleep-wake monitoring via sleep diaries and actigraphy, followed by one 8-hour laboratory visit during which salivary DLMO was assessed. DLMO and desired sleep times were used to classify participants' DSPD etiology as circadian (n=118; 61M; 28.8 ± 9.7 y) or non-circadian (n=67; 30M; 31.9 ± 12.2 y). Self-reported habitual bedtime on work nights was not significantly different between groups, (p=0.691), but a near significant trend was found toward later wake time in the circadian phenotype on work days (p=0.062). On non-work nights a near significant trend toward later bedtime in the non-circadian phenotype was shown (p=0.065, two-tailed), but the circadian phenotype woke significantly later on non-work days (p<.001). These results suggest that restrictions on sleep-wake behaviours engendered by the need to meet work/school obligations on work or school nights may mask the endogenous sleep-wake cycle, making it misleading in the diagnosis of DSPD. Assessing sleep-wake behaviour of DSPD patients on work and non-work nights separately may provide useful information but ultimately, measures of circadian phase are required to support a correct diagnosis and management of treatment.

Circadian tau differences in biological, behavioural and sleepiness rhythms in Delayed Sleep-Wake Phase Disorder and Non-24-hour Sleep-Wake Disorder patients.

Gorica Micic[1], Leon Lack[1], Nicole Lovato[1], Michael Gradisar[1], Sally A. Ferguson[2] and Helen J. Burgess[3]

[1]Flinders University of South Australia,
[2]Appleton Institute, Central Queensland University,
[3]Rush University Medical Center

Introduction: In this study we investigated sleepiness and behavioural rhythm period lengths (i.e., *taus*) of Delayed Sleep Phase Disorder (DSPD), Non-24-hour Sleep-Wake Disorder patients and healthy control sleepers. The aim was to explore if behavioural rhythms, in addition to the physiological circadian rhythms, may be contributing to misalignments of sleep timing symptomatic of DSPD and non-24.
Methods: Twenty-six DSPD participants who met diagnostic criteria (17m, 9f, age: 21.85±4.97 years) and 18 controls (10m, 8f, age: 23.72±5.10 years) participated in an 80-hour modified constant routine. Additionally, 4 full-sighted patients (3m, 1f, age: 25.75±4.99 years) were diagnosed with non-24 and included as a discrete study group. A forced-desynchrony ultradian protocol of 1-hour 'days' in dim light, controlled conditions alternated 20-minute sleep opportunities with 40-minute enforced wakefulness. Subjective sleepiness ratings were recorded prior to every sleep opportunity and median reaction time was measured hourly, 20-min into intervals of enforced wakefulness. Sleep onset latency was derived from 20-minute sleep opportunities to quantify hourly objective sleepiness. Rhythm data was curved using the 2-component cosine model.
Results: DSPD and non-24 patients had significantly longer melatonin and temperature taus compared to controls. Additionally, DSPD patients (M=24.42h±0.92h) had longer subjective sleepiness rhythm *taus* compared to the controls (M=23.81h±0.92, p=.03), and non-24 patients (M=24.00h±0.83h, p=.39), $F(2,47)=2.45$, p=.09. Differences between DSPD and controls were of a moderate effect size according to Cohen's criteria, d= 0.65. Furthermore, DSPD patients showed a longer interval between maximum and minimum feelings of sleepiness, while reaction speed and objective sleepiness rhythms did not show tau differences between groups.
Discussion: Feelings of sleepiness tend to build up differently and cycle significantly slower in DSPD patients compared to control sleepers and non-24. Results suggest this component could contribute to the sleep and thus biological rhythm delays in DSPD. Therefore, both cognitive and behavioural treatment approaches may be necessary for successful long-term outcomes.

DATA BLITZ ABSTRACTS

Deriving non-wear time thresholds for sleep/wake accelerometers based on patterns of bedtime quiescence

Anastasi Kosmadopoulos[1], David Darwent[1] and Gregory D. Roach[1]

[1]Appleton Institute, Central Queensland University, Adelaide, SA

Aims: The accuracy of sleep/wake estimates derived from wrist-actigraphy often depends on being able to discern non-wear from quiescent wakefulness or sleep. Non-wear could be inferred from periods of inactivity unlikely to occur during time in bed. The aim of this study was to develop an algorithm to estimate the likelihood of non-wear from bedtime quiescence.
Methods: Thirty-two participants lived in a sleep laboratory for 13 days. Scheduled bedtimes were equivalent to either 4h or 8h every 24h. Activity monitors recorded movement in 1-min epochs. The frequency of all consecutive zero epochs ≥1min during time in bed (TIB) were tabulated to derive a cumulative distribution function of inactivity.
Results: During TIB, there were 6,018 separated instances of inactivity ≥1 min, lasting an average of 14.7 (±18.5) min. Data collected suggest that, during TIB, 50% of inactive periods are <8 min, 75% of inactive periods are <18 min, and 95% of inactive periods are <53 min in duration.
Discussion: A 50% threshold, at which the device is more likely to be off than on, is probably sufficient for routine rest/activity monitoring. However, a higher threshold (e.g., 95%) may be more appropriate to discern non-wear around bedtimes where periods of inactivity are expected to be longer.

Glucose Metabolism is Impaired after One-Week of Sleep-Restricted Circadian Misalignment

Thomas Kontou[1], Gregory D. Roach[1] and Charlie Sargent[1]

[1]Appleton Institute, Central Queensland University, Adelaide, SA

Aim: The aim of this study was to investigate the combined effect of sleep restriction and circadian misalignment on glucose metabolism during sleep and to determine if this effect differed depending on sleep stage (NREM and REM).
Method: Nine healthy young males (mean age±SD: 20.6±2.3 years) underwent nine days of sleep-restricted forced desynchrony (4.7h time in bed and 23.3h wake, every 28 hours) that was preceded and followed by 8-hour sleep periods (0000h to 0800h). During 8-hour sleep periods, glucose metabolism was determined by measuring interstitial glucose concentrations using continuous glucose monitoring devices. Sleep was recorded using polysomnography.
Results: Interstitial glucose concentrations were significantly higher during the 8-hour sleep period after sleep-restricted forced desynchrony (mean±SD: 5.2±0.40mmol/L) than during the 8-hour sleep period before sleep-restricted forced desynchrony (mean±SD: 4.6±0.85mmol/L). Interstitial glucose concentrations did not differ between NREM and REM sleep and there was no interaction effect between study night and sleep stage.
Discussion: Glucose metabolism was impaired following sleep-restricted circadian misalignment but no differences in glucose metabolism were seen between sleep stages. In the absence of measures of insulin, it is not possible to determine if changes in glucose metabolism were due to reduced insulin sensitivity or impaired insulin secretion.

What is the relationship between social support, child sleep and Postnatal Depression?

Yaroslava King[1] and S Blunden[1]

[1]Appleton Institute, Central Queensland University, Adelaide, SA

Aims: To determine the extent to which social support and child sleep problems are affected by parental mood.
Methods: The sample consisted of 108 parents of children between 6-18 months of age. Participants completed an online survey comprised of The Edinburgh Postnatal Depression Scale, The Social Provisions Scale and The Brief Infant Sleep Questionnaire. Pearson's product-moment correlations and moderation analyses were utilised to explore relationships and moderation effects.
Results: Results showed significant relationships between PND and social support (r = -.539, p = .000), PND and nocturnal sleep (r = -.231, p = .016) and nocturnal wakefulness (r = -.228, p = .018). A simple slopes analysis revealed that when depression is high, there is a significant relationship between social support and nocturnal sleep (b = 4.936, 95% CI [0.040, 3.449], t = 2.03, p = .045).
Discussion: High scores of PND significantly moderate the relationship between parental social support and a child's nocturnal sleep. This indicates that social support is important to consider when investigating the factors associated with child sleep problems, particularly in depressed parents.

Too Hot, Too Tired: The Effects of Temperature and Sleep Restriction on Firefighters' Simulated Driving Performance

Raymond W. Matthews[1], Bradley P. Smith[1], Sarah M. Jay[1], Michael A. Cvirn[1], Sally A. Ferguson[1]

[1]Appleton Institute, Central Queensland University, Adelaide, Australia

This study aimed to investigate the effects of elevated temperature and sleep restriction on firefighters' driving performance, during a wildland fireground tour simulation. Twenty volunteer firefighter crew drivers (37.2 ±14.7 years of age, mean ± SD) underwent a multi-day firefighting simulation that involved real-world physical tasks, under either a control condition (23-25˚C daytime temperature and 8h sleep opportunities), an elevated temperature condition (33-35˚C daytime temperature), a sleep restriction condition (4h sleep opportunities), or a combined elevated temperature and sleep restriction condition. Driving performance was assessed by lane deviation on two daily 30min simulated drives replicating driving to and from the fireground. Mixed models analysis of variance revealed a significant main effect of condition on simulated driving performance [$F(3,111)= 6.86$ $p<.001$]. Driving performance in the combined elevated temperature and sleep restriction condition was significantly ($p<.05$) worse than all other conditions. There was also a non-significant trend such that in the elevated temperature condition, lane deviation was greater than control, and in the sleep restriction condition lane deviation was greater than both the elevated temperature and the control conditions. These results suggest that the combination of elevated temperatures and sleep restriction was associated with poorer driving outcomes than sleep restriction alone.

DATA BLITZ ABSTRACTS

Stroop performance after one night of sleep deprivation: Effect of BDNF genotype

Leilah Grant[1], Sean W. Cain[1,2]., Anne-Marie Chang[2], Charles Czeisler[2], & Clare Anderson[1,2]

[1] Sleep and Circadian Medicine Laboratory, Monash University
[2] Division of Sleep Medicine, Harvard Medical School and the Brigham and Women's Hospital

Accumulating evidence points to a genetic contribution to explain individual vulnerability to sleep loss. Recently the Brain Derived Neurotrophic Factor (BDNF) gene, which contains a functional polymorphism that causes a Valine (Val) to Methionine (Met) amino acid substitution, has become a gene of interest. Individuals with a Val/Met genotype are more at risk of cognitive dysfunction. Importantly these differences in cognitive outcomes can be exacerbated by a stressor such as sleep deprivation. Therefore, our aim was to investigate whether Met allele carriers are more vulnerable to the effects of executive dysfunction during sleep loss, as measured by the Stroop colour-naming task. Participants included 30 young healthy adults, 18 Val/Val homozygotes and 12 Val/Met heterozygotes. Participants were studied in a 30-hour constant routine protocol during which they remained awake in constant conditions and performed the Stoop colour-naming task every two hours. The version of the Stroop used for this study had three trial types- congruent (text and colour matched), incongruent (mismatch between text and colour) and neutral (coloured XXXXs) and required participants to name the colour of the text for each trial. For all three trial types Met allele carriers made more errors. For congruent and neutral trials both genotypes displayed similar reaction times. For incongruent trials, however, Met allele carriers took longer to respond than the Val/Val homozygotes. Our data suggests that Met allele carriers sacrificed speed for accuracy when required to inhibit the prepotent response of automatically reading the word. These results suggest that BDNF genotype may be an indicator of vulnerability to executive dysfunction during sleep loss.

During 50hrs of sleep deprivation caffeine decreases perceived sleepiness, however, does caffeine consumption alter hunger and satiety ratings?

Crystal Grant[1], J. Dorrian[1], A. Coates[2], GM. Paech[1], C. Della vedova[3], M. Pajcin[3], G. Kamimori, and S. Banks[1]

[1] Centre for Sleep Research, University of South Australia, Adelaide, SA, Australia
[2] Alliance for Research in Exercise, Nutrition and Activity, University of South Australia, Adelaide, SA, Australia
[3] School of Pharmacy and Medical Sciences, University of South Australia, Adelaide, SA, Australia

Introduction: Sustained operations not only involve sleep loss, but require individuals to remain awake during the biological night. The disruption of the sleep/wake cycle during sustained operations can be associated with sleep deprivation. Caffeine is a stimulant widely used as a countermeasure to decrease sleepiness. However, the impact of caffeine on hunger and satiety during sleep deprivation are largely unknown.
Methods: Participants were randomly assigned to either caffeine (n=12, 4F, 22.5±3.3y, 21.7±1.5kg/m^2) or placebo condition (n=12, 5F, 22.5±2.5y, 22.3±2.1kg/m^2). The in-laboratory protocol included one baseline sleep (22:00h–08:00h), 50h sleep deprivation, and one daytime recovery sleep (10:00h–19:00h). Caffeine (200mg) or placebo gum was administered at 01:00h, 03:00h, 05:00h and 07:00h during each night of sleep deprivation. Meal timing (07:10h, 13:00h, 19:00h and 01:10h) and composition were controlled. The KSS, hunger and satiety scales were administered at 10:00h, 16:30h, 22:30h and 04:30h each day.
Results: Participants in the caffeine condition reported significantly less sleepiness ($p<0.001$) throughout sleep deprivation. Hunger increased ($p<0.001$) and satiety decreased ($p=0.004$) with sleep deprivation. Caffeine did not moderate this effect (hunger $p=0.266$, satiety $p=0.770$).
Conclusions: Chewing caffeine gum did not affect hunger or satiety ratings, however caffeine was still an effective fatigue countermeasure.

Reducing Sleep-Onset Latency in Pre-School Children Using Slow-Frequency Tones: A Pilot Study

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The current pilot study examined the effects of rhythmic auditory stimulation in the form of 0.5-Hz tones on sleep-onset latency (SOL) problems pre-school aged children. Additionally, this study investigated whether there would be any secondary effects on anxiety (i.e., cognitive pre-sleep arousal) and problematic daytime behaviour (PDB; i.e., physiological pre-sleep arousal). Participants were one male 5-year-old and one female 3-year-old. Wrist actigraphy and a new proprietary bedside sleep monitor (Sense, Hello Inc.) were used to assess sleep quality as determined by SOL, total sleep duration (TSD) and night wakings (NW). Sleep and activity diaries completed by parents also provided subjective data regarding the childs' SOL, TSD, and NW. Anxiety was assessed by parents using the Spence Children's Anxiety Scale (Spence, 1998) and PDB was recorded by parents using a PDB diary. Exploratory case study analyses were conducted. Slow-frequency tones appeared to reduce SOL and anxiety in each participant. The effect of slow-frequency tones on PDB was inconclusive. Additional empirical group-based research is needed to further investigate the effect of slow-frequency tones on SOL, anxiety, and PDB.

A pilot study investigating the effectiveness of listening to a metronome sound that can help children with autism to initiate sleep

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To investigate the effectiveness of listening to a rhythmic auditory stimulus that can reduce sleep onset latency (SOL) among children with autism. The present study aimed to measure the validity of the newly developed sleep monitor, (Sense, Hello Inc) relative to writs actigraphy (wGT3X-BT Monitor, Actigrphay Pty Ltd) data in measuring sleep/wake patterns of children with autism. It was hypothesised that less night-awakenings, shorter SOL and sleep duration, and lower parental anxiety scores are found when children listened to intervention metronome sound during bedtime than no sound intervention and white sound. A total of four children with autism (M = 4.75 years, SD = 0.96 years) participated a study who received no sound intervention (baseline week) during bedtime; then randomly allocated to receive either intervention or white sound for a week on the following week, and receiving the alternative sound during the third week. Parents completed an anxiety questionnaire, and sleep- and problem behaviour-diaries were also recorded throughout the study. Results indicated that Sense sleep monitor did not provide sleep data consistent with actigraphy and sleep diary data. No sleep variables and anxiety differences were observed across the three-study weeks. Replication of the current study with a larger sample is required to investigate whether rhythmic auditory stimulus is a practical intervention for helping children with autism to fall asleep faster.

DATA BLITZ ABSTRACTS

Panax Ginseng as an adjunct to the Continuous Positive Airway Pressure therapy in the management of Obstructive Sleep Apnoea

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Although Continuous Positive Airway Pressure (CPAP) therapy is the most effective treatment available for Obstructive Sleep Apnoea (OSA), compliance remains poor. The aim of this study was to investigate the effectiveness of Panax Ginseng as an adjunct to CPAP therapy for the treatment in individuals with OSA. It was hypothesized that Panax Ginseng would have positive effects for OSA patients, by reducing nasal inflammation, improving sleep quality, cognitive performance and daytime alertness. Participants: sample size was 9 with 8 males and 1 female aged between 39 to 70. There were 3 sessions ran for a period of 5 weeks. Participants were randomized into two groups: an experimental group (Panax Ginseng) or a placebo control group for 2 weeks, with 1-week washout in between. Outcome measures included upper-airway inflammation (nitric oxide in breath condensate, NOSE Questionnaire), cognitive performance (Subtle Cognitive Impairment Test (SCIT); Psychomotor Vigilance Task, (PVT)), and sleep quality (derived from wrist actigraphy and S+ bedside monitor, Epworth Sleepiness Scale (ESS)).

Antidepressant medication increases the response of the circadian system to light

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Abnormalities of the circadian system are often observed in patients with depression. In animal models, selective serotonin reuptake inhibitors (SSRIs), the most common antidepressant, significantly alter the responsivity of the circadian system to light. We examined the effect that a single dose of the SSRI Citalopram (30mg), has on the sensitivity of the circadian system in a group of healthy men. The trial had a between-subjects, placebo-controlled, counterbalanced design. Participants were required to maintain a consistent sleep-wake schedule for a minimum of one week prior to each of three test sessions, which occurred one week apart. Salivary melatonin was measured in each of the test sessions, with the first serving as a dim-light baseline. During the subsequent two test sessions participants were exposed to lighting of ~350 lux and took either a single dose of Citalopram or a placebo on each occasion. Melatonin suppression was calculated by comparing each of the active conditions (placebo or Citalopram with light) to the dim-light baseline. Twice the level of melatonin suppression was observed in the Citalopram condition, indicating a significant increase in the sensitivity of the circadian system. This may result in normalisation of rhythms in some patients, while causing further disruption in others.

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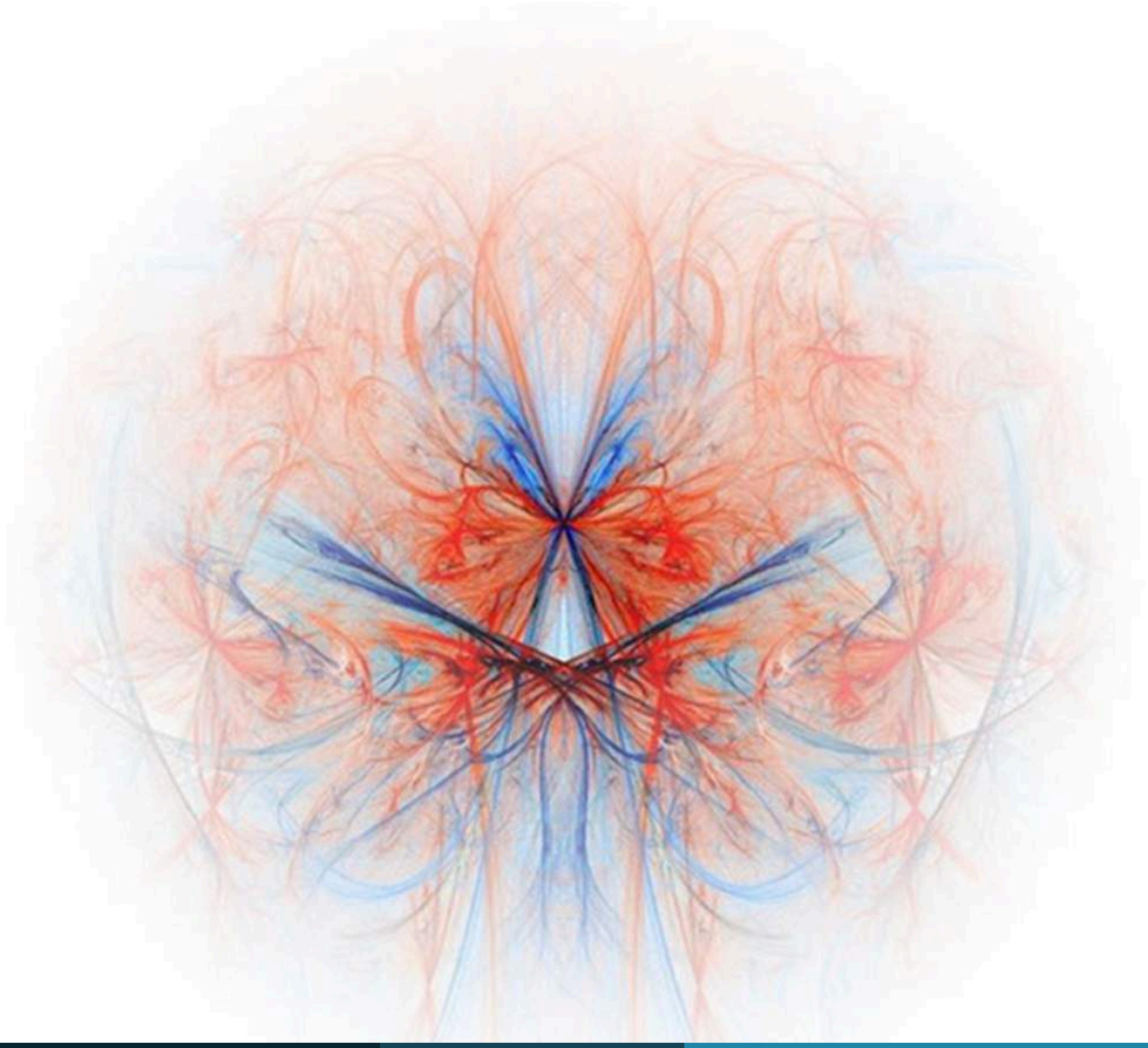

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