

# Crown court

## Executive summary

X was assessed on the 27.05.2015 in HMP Bristol, from 14:40 hours to 16:00 hours, a period of an hour and twenty minutes. He had a depressive mental state as a consequence of his physical ill-health and as a consequence of his most recent arrest and remand on charges of a “conspiracy to manufacture and supply class A and B controlled drugs to another”. He had an age and ill- health related cognitive impairment (the early stages of dementia). His complaints of memory problems appeared genuine and related to his carcinoma of the prostate and pharmaceutical treatment thereof, improperly treated hypertensive state (high blood pressure) and associated vitamin D and folic acid deficiencies. From the information provided, and from assessment, he understood the charges levied against him and the consequences of conviction. As a consequence of his blunted affective disorder (depressed mental state) it was believed that he would have difficulty in comprehending and retaining all of the advanced degrees of information required in examination and cross-examination, because of his short attention span. It was believed that he would be unfit to plead and unfit to stand trial in his current mental state. That if he did stand trial any available measures such as assistance, frequent breaks and modifications to the manner of questioning, would assist his comprehension of events.

## Issues to be addressed I

Provision of an expert opinion on the following specific areas:

- i. X’s current mental state
- ii. X’s possible mental state at the time of the alleged offences
- iii. Issues of diagnosis and possible treatments
- iv. Fitness to plead and stand trial
- v. The possibility of the defendant feigning injury
- vi. Whether the defendant’s degree of cognitive impairment was such as to place in doubt his ability to advise his solicitors in relation to the details of the evidence which can reasonably be expected to be given in his case, as his memory for the detail will be impaired.
- vii. Whether the above condition is likely to hamper his ability to instruct his legal advisors before or during trial and in turn their ability to conduct a proper defence.
- viii. Whether the above is likely to affect the defendant’s ability to give evidence in his own defence, whether his difficulty in remembering would impair him to give evidence, whether this could be compounded by difficulties in retaining questions that he was asked and in applying his mind to answering specific questions
- ix. Whether his tendency to forget what he is talking about in mid-sentence would be likely to be exacerbated by the stress of the court setting.
- x. Whether the defendant’s difficulties were too severe to be overcome by special measures such as assistance, frequent breaks and modifications to the manner of questioning.

Volume 2 Issue 3 - 2016

Gu Y, I Pates J,<sup>2</sup> Graham MR<sup>1</sup>

<sup>1</sup>Research Academy of Grand Health, Ningbo University, China

<sup>2</sup>Llantarnam Research Academy, UK

**Correspondence:** Graham MR, Ningbo University, Zhejiang, 315211, Llantarnam Research Academy, Newport Road, Cwmbran, Torfaen, NP44 3AF, China, Tel 01633 483166, Email [professormrgraham@advocacy.london](mailto:professormrgraham@advocacy.london)

**Received:** February 24, 2016 | **Published:** May 2, 2016

**The history below was provided from the documents made available by solicitors, & by the defendant, during an interview on Xx.Xx.2015, 14:40-16:00 hours**

## Personal history

The defendant had received a private education. He finished full-time education at the age of 15 years. The defendant was divorced ten years previously for drug offences, which compromised his relationship with his wife and children. His youngest child a daughter, was aged 17 years and a son, had visited him in prison several times. The defendant was a non-smoker, had not abused alcohol and had not used recreational drugs.

## Forensic history

The defendant had multiple convictions, including fraud, theft and drug offences, since 1960. He had received multiple custodial sentences, the first was in 1961.

## Alleged index offence

The prosecution allegation centred on an illicit drugs production and manufacturing operation that was being set up by an organised crime gang (OCG). The prosecution allegation is that to fund the set-up of the production and manufacturing operation, the OCG, including the defendant were involved in the manufacture and supply of Class A and Class B drugs. The prosecution case is based on surveillance techniques, CCTV, mobile phone device analysis and audio tape.

## Past & current medical history

The defendant was diagnosed with Hypertension ten years previously, which was only treated by his general practitioner in 2009. He often used to forget to take his medication, suggesting that his hypertension had not been appropriately treated. The defendant was diagnosed with prostate cancer, in May 2013 and had received multiple courses of radiotherapy and was receiving the medication goserelin (Zoladex) which is an anti-testosterone hormone injection. He was experienced side effects from this injection, including the loss of body hair, growth of breast tissue, absent libido and weight gain.<sup>1</sup> The defendant required to urinate up to ten times every night, causing sleep disturbance. There was no toilet in his cell, which was

a contravention of his human rights. The defendant experienced significant gastric side effects from goserelin injections and that he continually experienced a bloated gaseous abdomen. The defendant had a computerised tomogram (CT scan) of his brain on XX.XX.2015. The defendant had a low vitamin D level.

### Medical history

Skeletal MRI scan report dated XX.XX.2014 showed no skeletal metastases (secondary cancer). \*Results demonstrated he had Metabolic Syndrome, a disorder of energy utilization and storage, diagnosed by a co-occurrence of three out of five of the following medical conditions:

- i. Abdominal (central) obesity;
- ii. Elevated blood pressure (Hypertension);
- iii. Elevated fasting plasma glucose;
- iv. High serum triglycerides;
- v. Low high-density lipoprotein (HDL) levels.

The defendant suffered with:

- i. Obesity
- ii. Dyslipidaemia: [High total Cholesterol (TC): 6.1 mmol/L {<5.2}; high Triglycerides (TG): 3.5 mmol/L {<2.1}; high Low Density Lipoprotein (LDL): 3.4 mmol/L {<3.0}; low High Density Lipoprotein (HDL): 1.1 mmol/L {>1.2}; TC/HDL ratio: 5.5 mmol/mmol (<3.5);
- iii. Hypertension: [Grade 3] 100/60 mm. Hg, February, 2015; 156/96 mm. Hg, March, 2014; 224/94 mm. Hg, January 2010.
- iv. Insulin Resistance: Haemoglobin (Hb)A1c 41 mmol/mol (6.5-7.5% = 48-59 mmol/mol)
- v. Non diabetics, reference range = 20-41 mmol/mol (4%-5.9%)
- vi. HbA1c levels between 5.7% and 6.4% indicate increased risk of diabetes (pre-diabetes).
- vii. Metabolic Syndrome is an independent risk factor for neurological disorder, including Alzheimer's syndrome.<sup>2</sup> The defendant's serum lipid levels were reported in 2014 as "Abnormal. No further action."

These abnormalities should have been treated with lipid lowering medication.

- i. Disordered electrolyte imbalance: Potassium 5.4 mmol/L, February 2015 (Range=3.5-5.3 mmol/L) which is a known cause of memory dysfunction.
- ii. Thyroid function: Tetra-iodothyronine (T4) is low normal (15.7 pmol/L [Range=12.0-22.0 pmol/L]. Tri-iodothyronine (T3) is a known cause of memory dysfunction has not been measured and assessed. There is no explanation as to why this has not been performed?
- iii. Renal dysfunction: Sustained Elevated Creatinine 146 umol/L (Range=60-110 umol/L) Urea has not been measured and assessed and is a known cause of memory dysfunction. There is no explanation as to why this has not been performed?

- iv. Testosterone low (<0.3 nmol/L [8.7-29]) and Luteinising Hormone (LH) <0.5 IU/L (1.6-9.6) are both very low and are known causes of memory dysfunction.
- v. Haemoglobin had dropped from 16.4 g/dL (November, 2011) to 13.5 g/dL (April, 2015) (Range=13.0 – 17.0 g/dL) and Packed Cell volume had dropped from 0.49 to 0.42 (Range=0.40-0.52) in 48 months (equivalent to losing 2 pints of blood) and are known causes of memory dysfunction.
- vi. March, 2015: Low serum Albumin (protein) 34 g/L (Range=35-50 g/L) suggested early liver dysfunction and is a known cause of memory dysfunction.
- vii. March, 2015: Low vitamin D (19 nmol/L [<25 nmol/L=severe deficiency]) and Low folate (3.2 ug/L [Range=4.20 - 18.70 ug/L]).<sup>3,4</sup> Both are associated with depression and memory dysfunction.

These results all indicated that the defendant had a genuine medical condition at the bio-molecular, neurochemical level, to account for both his depressive mental state and his cognitive decline.

### Past psychiatric history

The defendant had never seen his general practitioner, or a psychiatrist nor a mental health specialist in the community for memory loss, until admission to prison. Since his arrest he had continuous thoughts and feelings of sadness worthlessness, helplessness, hopelessness and the futility of life (ruminations). He worried continuously about his children. He denied being suicidal, but every night he went to sleep wishing that he would not wake up. On multiple occasions during a normal day he admitted wishing that he was dead. He did not have difficulty going to sleep, because in the prison he was prescribed amitriptyline as a sleeping medication, but he continuously awoke to urinate and had early morning waking and then could not get back to sleep. He spent most of his time in isolation in his cell. Mr Rogers was currently prescribed esomeprazole (for dyspepsia), mebeverine and peppermint oil (for abdominal cramps), tamsulosin (for enlarged prostate), cholecalciferol (vitamin D supplement), goserelin injections (hormone treatment for prostate cancer), ramipril (for high blood pressure), paracetamol, amitriptyline (low dose antidepressant helping sleep) and cetirizine (for allergies)

### Interview

The interview lasted for one hour and twenty minutes. The defendant was aware that the expert had been asked to assess his memory by his legal team. He provided three (3) hand written pages of his current health complaints and latest results. He described his memory problems and that his memory had been getting worse. He described events from many years ago, concerning his family. He described not wanting visitors as he was ashamed of being in prison. He stated that all he could do in prison was watch television, that he did not have the attention span or concentration to read a book. He remembered watching the programme "Churchill: When Britain said 'No'" and was able to offer an opinion on it. He stated that he was wary of other prisoners that they might take things from his cell. He was in a single cell, but did not have a toilet and at night was provided with a bottle, which made urinating very difficult. He did not have a "job" (occupation) in prison, due to Health and Safety regulations, because of his age and his health status. He stated that in Court he

struggled to follow proceedings. He stated that his charges were as a consequence of him being recorded talking about drugs, with his co-defendants, which was spurious. He stated that he had heard his conversations and his language was foul and abusive, but that he did not normally talk in such a fashion.

**Mental state examination**

The defendant was dressed in prison garb and unshaven. He did not maintain good eye contact but stared down at the floor. Levels of psychomotor activity were retarded. His speech was not spontaneous, quiet and sometimes difficult to comprehend, requiring to be repeated. He appeared dysthymic in mood. In his history there may have been evidence of delusional thought content when he talked of being in possession of millions of pounds of bearer bonds and property equity, which had been confiscated years before. There was obsessional thought content in relation to his children, but no evidence of current, nor past hallucination. His cognitive state was examined in detail below.

**Hospital anxiety and depression scale (HADS) questionnaire**

More than 200 published studies worldwide have reported experiences with the Hospital Anxiety and Depression Scale (HADS) questionnaire<sup>5-8</sup> for use with physically ill patients. The questionnaire consists of 14 questions: 7 questions are related to anxiety and 7 questions are related to depression. Each item is rated from a score of 0 to 3, depending on the severity of the problem described in each question, giving a maximum subscale score of 21 for anxiety and depression, respectively. The anxiety and depression scores are categorised in (Table 1) below.

**Table 1** Hospital anxiety and depression scale questionnaire scores

Aggregate score	Interpretation
0-7	Normal
08-Oct	Mild
Nov-14	Moderate
15-21	Severe

The HADS gives clinically meaningful results as a psychological screening tool in correlational studies with several aspects of disease and quality of life. It is sensitive to changes both during the course of diseases and in response to psychotherapeutic and psychopharmacological intervention. HADS scores predict psychosocial and possibly physical outcome.<sup>9</sup> This self-assessment scale was originally developed and found to be a reliable instrument for detecting states of depression and anxiety in the setting of a hospital medical outpatient clinic. The anxiety and depressive subscales are also valid measures of severity of emotional disorders. The defendant scored 16/21 for both Anxiety and Depression, which is severe.

**The hamilton depression scale questionnaire (HAMD)**

There was current evidence of active depression. The Hamilton Depression Scale questionnaire (HAMD), gave a score of 37/66, which was abnormal and high.<sup>10</sup> This correlated with the HADS.

**The buss-durkee hostility inventory (BDHI)**

The ‘Buss-Durkee Inventory’ on feelings of hostility/aggression questionnaire was normal.<sup>11</sup>

**Psychometric testing**

Defendant completed the Addenbrookes Cognitive Examination-III (ACE-III) this replaced the ACE version and ACE-R version 2005 in November 2012. The ACE-III is an established screening test for dementia, with the participant completing a range of tests in different cognitive domains and being given a score out of 100. Two cut offs are used to identify ‘positive’ results (i.e. cognitively impaired); 88 and 82. The defendant was encouraged during the test and provided his best effort. In order to check the consistency of his responses, the defendant’s previous scores on the ACE-R completed one month earlier 08.04.2015. Results are below (Table 2).

**Table 2** Defendant’s previous scores on the ACE-R

Domain	Score on XX.05.2015	Score on XX.04.2015
Attention and orientation	13/18	Dec-18
Memory	16/26	Dec-26
Fluency	Mar-14	Mar-14
Language	19/26	15/26
Visuospatial	14/16	14/26
Total	65/100	56/100

These results were consistent and indicated the presence of cognitive impairment and were suggestive of the defendant suffering with dementia. Contained within the ACE-III is the Mini Mental State Examination (MMSE), another test for cognitive impairment. The MMSE is probably the most commonly used test for memory problems. A score of 25 or above is considered decreased odds for dementia. The defendant scored 26/30 on the MMSE, which is borderline for dementia. The coin test gave result of five (5) out of ten (10) suggesting malingering.<sup>12</sup> The defendant was unable to complete the Test of Memory and Malingering (TOMM) because of interview time limitation.<sup>13</sup>

**Physical examination**

Body weight: 19 stone (120.9 kg)

Height: 6 foot (180 cm)

Body Mass Index (BMI): 37.3 kg m<sup>-2</sup>. (Severe to Morbidly obese) (Table 3).

**Table 3** The International classification of adult underweight, overweight and obesity values according to BMI

Classification	BMI (kg/m <sup>2</sup> )
Principal values	
Obese	≥30.00
Obese class I	30.00 - 34.99
Obese class II	35.00 - 39.99
Obese class III	≥40.00

The defendant’s CT Brain scan on 30.04.2015, is reported as: “No focal intraparenchymal mass lesion identified. No haemorrhage or surface collection seen. Moderate generalised involutational change but with no particular focal atrophic element”.

This is indicative of age related global decline.

## Diagnosis, opinion and recommendation

The tests clearly indicated that the defendant had an active undiagnosed and untreated depressive mental health state. He had a cognitively impaired mental health state (mild dementia). There had been a progressive decline in memory primarily affecting short term recall, which is expected in a man of his age, but had been compounded by his carcinoma of the prostate, pharmaceutical treatment and his intermittently treated hypertension (high blood pressure) and is suggestive of the early stages of dementia. This was correlated by the results of his CT scan. There was no evidence of a severe and enduring mental illness (e.g. schizophrenia). There was evidence of a depressive mental illness. There was a suggestion of a personality disorder, a Walter Mitty type personality disorder with mild delusional state.<sup>14</sup> The defendant suffered with prostate cancer and hypertension, which are causative of cognitive decline. Prostatic carcinoma is known to metastasise to the brain and cause dementia. Hypertension is the commonest vasculopathy known to cause dementia. He denied using drugs, and appeared to be in the normal range of intelligence. His performances on cognitive testing, indicated a diagnosis of early dementia compounded by his blunted affective disorder (depressed mental state). More detailed imaging investigations, magnetic resonance imaging (MRI) or positron emission tomography (PET) would identify if there were any focal lesions accounting for his dementia and to exclude or confirm if he had metastatic carcinoma.

## Issues to be addressed 2

The defendant's performance on affective mental state is cognitive testing.

- i. *The defendant's current mental state:* This is dealt with in detail above.
- ii. *The defendant's likely mental state at the time of the alleged offences:* The alleged offences took place in late 2013 to early 2014. The defendant's own account was that his memory had deteriorated since this time. If he were suffering from any cognitive impairment at the time this would have had an impact on his behaviour. He may have become mildly delusional and developed a Walter Mitty type personality type disorder, where one imagines one is successful and popular, instead of making real efforts to make friends and succeed at a job'. Fantasy, when pushed to the extreme, is a common trait of narcissistic personality disorder; and not one person who uses fantasy a lot has any close friends'. He may have not retained the capacity to make decisions about his behaviour.<sup>14</sup>
- iii. *Issues of diagnosis and possible treatments:* Tests such as MRI or PET scan and vitamin and mineral screen and full analysis of lipoproteins and inflammatory markers were required for a definitive diagnosis. Treatment for depression with a modern day serotonin and noradrenaline reuptake inhibitor (SNRIS) and management of urinary and bowel conditions is mandatory. Should his cognitive ability deteriorate, enabling a causation for dementia to be made, then he should be treated according to NICE guidance, including medication which may slow down (although not reverse) any cognitive decline, such as cholinesterase inhibitor, donepezil.<sup>1</sup>
- iv. *Fitness to plead and stand trial:* The defendant's fitness to plead was a crucial area. He understood the charges against him. He understood the difference between a guilty and not guilty plea. He understood the concept of challenging a juror, and described a good understanding of Court processes. The more relevant areas were his ability to follow evidence in Court and ability to give appropriate legal instruction. He described that, as a result of having multiple co-defendants, proceedings had become confusing. He described being unable to follow what was happening when anyone spoke. The case against him was complex, with the number of co-defendants and reliance on audio recorded evidence. Any degree of cognitive impairment, made it more difficult to follow such evidence in Court. He had extreme difficulty following evidence in Court and providing appropriate instruction to his legal representatives. It would have been an absolute necessity to provide special measures to him, including frequent breaks, use of simplified language and assistance when being questioned.
- v. *The possibility of the defendant feigning injury:* During assessment he performed as well as he could under the circumstances. In view of his performance in the cognitive assessments it was not possibly to say whether he did have an underlying, much severe form of cognitive impairment. The coin test suggested malingerer, but was inconclusive.
- vi. Whether the defendant's degree of cognitive impairment was such as to place in doubt his ability to advise his solicitors in relation to the details of the evidence which could reasonably be expected to be given in his case, as his memory for the detail would be impaired. It was considered that he did not have the capability of recalling specific details about events which took place relevant to the case against him and advising his representatives appropriately.
- vii. Whether the above was likely to affect the defendant's ability to give evidence in his own defence, whether his difficulty in remembering would impair him to give evidence, whether this could be compounded by difficulties in retaining questions that he was asked and in applying his mind to answering specific questions. It was considered that it would be helpful to take steps of offering individual support, frequent breaks in proceedings and questions being asked using simplified language to assist him in giving evidence. It was considered that his cognitive state would prevent him giving satisfactory evidence in his own defence and that he should not give evidence.
- viii. Whether his tendency to forget what he is talking about in mid-sentence would be likely to be exacerbated by the stress of the court setting. Losing train of thought mid-sentence is powerfully associated with cognitive impairment. It was considered that there were periods of distraction and long pauses before answering which were as a consequence of his low mood state. The stress of court would have had a negative impact on his ability to concentrate. It was considered that he could raise such impairment as part of his defence and unfitness to plead.
- ix. Whether the defendant's difficulties were too severe to be overcome by special measures such as assistance, frequent breaks and modifications to the manner of questioning. The

defendant's difficulties were severe enough to make him unfit to plead and he should not provide evidence.<sup>15–30</sup>

## Conclusion

The defendant did not give evidence. He was convicted of the alleged charges. He was sentenced to 18 years.

## Acknowledgments

None.

## Conflicts of interest

The author declares that there are no conflicts of interest.

## References

1. British National Formulary (BNF). 2016.
2. Farooqui AA, Farooqui T, Panza F, et al. Metabolic syndrome as a risk factor for neurological disorders. *Cell Mol Life Sci.* 2012;69(5):741–762.
3. Thomas NE, Baker JS, Graham MR, et al. C-reactive protein and its relation to adiposity, physical activity, aerobic fitness and habitual diet. *Br J Sports Med.* 2008;42(5):357–360.
4. Thomas NE, Cooper SM, Baker JS, et al. Homocysteine, folate, and vitamin status. *Res Sports Med.* 2008;16(4):233–243.
5. Graham MR, Davies B, Evans P, et al. Growth Hormone therapy in health and disease. Could GH & IGF-I combination therapy combat the somatopause? *Journal of Exercise Physiology.* 2009;12(6):1–24.
6. Graham MR, Evans P, Davies B, et al. Arterial pulse wave velocity, inflammatory markers, pathological GH and IGF states, cardiovascular and cerebrovascular disease. *Vasc Health Risk Manag.* 2008;4(6):1361–1371.
7. Graham MR, Davies B, Hullin D, et al. Recombinant human growth hormone in abstinent androgenic-anabolic steroid use: Psychological, endocrine, and trophic factor effects. *Curr Neurovasc Res.* 2007;4(1):9–18.
8. Zigmond AS, Snaith RP. The hospital anxiety and depression scale. *Acta Psychiatr Scand.* 1983;67(6):361–370.
9. Herrmann C. International experiences with the Hospital Anxiety and Depression Scale, a review of validation data and clinical results. *J Psychosom Res.* 1997;42(1):17–41.
10. Hamilton M. A rating scale for depression. *J Neurol Neurosurg Psychiatry.* 1960;23:56–62.
11. Buss AH, Durkee A. An inventory for assessing different kinds of hostility. *J Consult Psychol.* 1957;21(4):343–349.
12. Kapur N. The coin-in-the-hand test: a new “bed-side” test for the detection of malingering in patients with suspected memory disorder. *J Neurol Neurosurg Psychiatry.* 1994;57(3):385–386.
13. Tombaugh NT. *Test of Memory and Malingering.* MHS. 1996.
14. Airagnes G, Consoli SM. Organic delusional states. *Rev Prat.* 2015;65(2):241–243.
15. Mioshi E, Dawson K, Mitchell J, et al. The Addenbrookes Cognitive Examination Revised (ACE-R): a brief cognitive test battery for dementia screening. *Int J Geriatr Psychiatry.* 2006;21(11):1078–1085.
16. <http://www.neura.edu.au/frontier/research/>
17. <http://www.gla.ac.uk/schools/medicine/>
18. Graham MR, Davies B, Baker JS. Causes and Consequences of Obesity, Epigenetics or Hypokinesia? *Diabetes Metab Syndr Obes.* 2015;8:455–460.
19. Graham MR, Davies B, Baker JS. *PIIINP a biomarker of endothelial dysfunction.* Oxidative Medicine and Cellular Longevity, In Press. 2015.
20. Graham MR, Baker JS, Davies B. *Peptide Hormones, and new-wave practices and research therapies.* Palgrave Macmillan Publishers, London, UK. 2015.
21. Graham MR, Baker JS, Davies B. *Obesity and alternative anorectics.* Palgrave Macmillan Publishers, London, UK. 2016.
22. Graham MR, Davies B, Baker JS. Can we Learn from Past Iatrogenic Effects When Managing Obesity? *J Sports Med Stud.* 2015;5:1.
23. Graham MR, Davies B, Evans PJ, et al. Exercise Science: Traditional and Emerging Trends. *J Sports Med Doping Stud.* 2012;2(3):1–9.
24. Graham MR, Evans P, Thomas NE, et al. Changes in endothelial dysfunction and associated cardiovascular disease morbidity markers in GH-IGF axis pathology. *Am J Cardiovasc Drugs.* 2009;9(6):371–381.
25. Baker JS, Graham MR, Thomas N, et al. Are we significantly underestimating the quantification of anaerobic performance in overweight and obese subjects? *The Open Sports Medicine Journal.* 2008;2:14–15.
26. Graham MR, Davies B, Evans PJ, et al. *Endothelial function, lipid profile, RPP & PIIIP, Microvascular and endothelial Physiology.* The Physiological Society's Cross Themed Meeting, England, UK. 2010.
27. Graham MR, Ryan P, Evans PJ, et al. *Insulin resistance Sport, Lipid profile, packed cell volume & BMI.* King's College London. Proceedings of Physiological Society, UK. 2009.
28. Graham MR, Baker JS, Kicman A, et al. *Cardiovascular Profiles.* Wales Virtual Institute of Sport Health & Exercise Science (WISHES) 5th annual conference, UK. 2008.
29. Graham MR, Baker JS, Davies B. *Keynote presentation. Hypertension and memory loss.* BASES Student Conference, University of Bedford, UK. 2008.
30. Azme S, Kate W, Shai M, et al. The Hamilton Rating Scale for Depression: a comprehensive review. *J Operational Psychiatry.* 1979;10:187–190.