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Our Reference: PFTDC.26.14 **Your Reference: Oury Jalloh**

FORENSIC TOXICOLOGY REPORT

By

Michael Scott-Ham B Sc (Hons.), MCSFS

Concerning

The death of Oury JALLOH

Date of Report: 18th June 2015

Report prepared for the Court on the instructions of:

Nadine Saeed

Initiative in Remembrance of Oury Jalloh, e.V., Germany

Principal Forensic Services

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Qualifications and Experience

I hold an Honours Degree in Applied Chemistry and work as a Forensic Consultant. From 1978 to March 1996 I was employed at the Metropolitan Police Forensic Science Laboratory, London (MPFSL) and this employment continued when the laboratory merged with the United Kingdom's Forensic Science Service (FSS). I left the Forensic Science Service at the end of February 2012 when the service was closed down. Whilst there, from 2006 onwards, I was the Principal Scientist for Toxicology.

I have more than 20 years of experience in the field of Forensic Toxicology and 10 years in the field of Forensic Drug Analysis. Prior to closure of the FSS I was a United Kingdom Home Office Authorised Analyst for the purposes of the Road Traffic Act for more than 20 years.

I am a committee member of The United Kingdom and Ireland Association of Forensic Toxicologists (UKIAFT), a member of The International Association of Forensic Toxicologists (TIAFT), the Society of Forensic Toxicologists (SOFT) and LTG (formerly London Toxicology group). I am also a Senior Associate member of the Royal Society of Medicine and a Professional Member of The Chartered Society of Forensic Sciences.

I have peer-reviewed scientific papers for Science and Justice, the Journal of Forensic and Legal Medicine and Forensic Science International and have recently (2013) had a peer-reviewed paper entitled "Concentrations of drugs determined in blood samples collected from suspected drugged drivers in England and Wales" published in the Journal of Forensic and Legal Medicine.

I have recently (September 2014) had a book published "Symptoms and Signs of Substance Misuse" (CRC Press) for which I was a co-author.

Since April 2013 I have been advising the United Kingdom Home Office and Department for Transport regarding drugs and driving via an Expert panel and more recently as an independent consultant.

Instruction

I have been requested to comment on the toxicology reports relating to the death of Oury Jalloh in January 2005.

My report is based on the information seen; should this information change, or further information become available, it may be necessary to reassess my findings.

This report has been peer-reviewed.

Enclosures

A large number of photographs and other documentation has been supplied but only those with a direct relevance to toxicology have been referred to and are listed below:-

- Autopsy Report by Professor Kleiber (translation); a part of the original German document has also been referred to
- Autopsy Report by Professors Bratzke and Kauert (translation)
- Forensic Medical Report by Professor Bohnert (translation)

Relevant Information

From the information supplied I understand that Oury Jalloh was arrested by police on the morning of the 7th January 2005 after allegedly making a nuisance of himself to passers-by. Due to his behaviour he was placed in a 'sobering-up' cell and was fixed to a bed, covered by a mattress, with hands and feet immobilised via hand and foot-cuffs. The cell was allegedly checked several times, the last time being around 11.45am. Just after midday a fire alarm activated having been caused by a fire in Oury Jalloh's cell. It was reported that the smoke was too dense for police to enter the cell and fire services were called. When the cell was entered Oury Jalloh was found to be dead with severe burns and still in cuffs.

A post-mortem examination (autopsy) was commenced at 8.25pm the same day and concluded at 10.30pm.

No carbon monoxide was detected (this would have been via analysis as carboxyhaemoglobin COHb). Soot was found in the airways and a small amount in the stomach.

Samples taken at the examination for possible toxicological examination included liver, kidney, brain, gall bladder, stomach, duodenum, urine and blood/serum. I was unable to find any reference to what site in the body the analysed post-mortem blood/serum sample had been taken from.

A blood sample had been taken at around 9.15am, which was prior to Oury Jalloh's detention, and alcohol was detected within this sample.

Toxicology Analysis

In Professor Kleiber's report it states that 2ml of blood, 3ml of serum, 3ml of urine and 5g of liver were examined.

Analysis for alcohol was performed via use of headspace gas chromatography, probably using flame-ionisation detection (GC-FID).

The urine sample was screened for cannabis, opiates, cocaine and amphetamines using immunoassay.

Basic and acidic screening for a range of drugs was performed on the liver and urine samples using HPLC (high-pressure liquid chromatography) and quantification of any drugs present was carried out using GC-MS (gas chromatography-mass spectrometry).

All of these are standard methods for such analyses.

The following results were reported in Professor Kleiber's report:-

Drug	Serum	Urine
THC-carboxylic acid (or THC-acid)	<10ug/l	15.0ug/l
Cocaine	<10ug/l	1.4mg/l
Ecgoninemethylester*	54.8ug/l; 42.2ng/ml**	About 6.7mg/l
Benzoylecgonine	171.6ug/l; 142.7ng/ml**	About 10.7mg/l

No medication was detected in the urine or liver samples.

Cyanide (CN) and carboxyhaemoglobin (CO-Hb) "could not be proved".

*There appears to be some confusion as to what compound was detected as both ecgoninemethylester (Professor Kleiber's report in German) and ecgonineethylester have been reported as well as ecgoninmetylphenidate (translation from Professor Kleiber's report).

[Please note that ecgoninmetylphenidate must be an incorrect translation as no such compound exists].

** Two different results were reported for each compound, one for a sample labelled "10/05" and another labelled "98/05".

No carbon monoxide was detected in the post-mortem blood sample.

The early (ante-mortem) blood sample, taken around 9.15am, was reported to contain alcohol at a concentration of 2.98% (= 2.98 milligrams per gram).

The autopsy blood sample was found to contain alcohol at a concentration of 2.68 milligrams per gram.

The autopsy urine sample was found to contain alcohol at a concentration of 3.42 milligrams per gram.

Further samples were taken on the 31st March 2005 at another autopsy and included a "serosanguinous" sample squeezed from the lung (no blood sample was available). A similar sample was obtained via squeezing of the liver. Brain tissue was also taken.

All of these samples were subjected to toxicological examination.

Basic and acidic screening for a range of drugs was performed on the serosanguinous lung sample using GC-MS with derivatisation. Analysis for cyanide and alcohol was also performed as well as analysis for volatile substances, again by GC-MS.

Cyanide and carboxyhaemoglobin were tested for on the serosanguinous liver sample, the latter via a photometric method. The cyanide determinations were via the Conway diffusion procedure.

Volatile substances and alcohol were tested for in the brain tissue using GC-MS.

The following results were reported by Professors Bratzke and Kauert:-

Drug	**Lung**	**Liver**	**Brain**
Benzoylecgonine	0.17mg/l		
Ecgonineethylester	0.06mg/l		
Caffeine	Low		
Cyanide	0.56mg/l	0.12mg/l	
Ethanol	Detected (NQ)		Detected (NQ)
Volatile substances	Not detected		Not detected
COHb		Approximately 6%	
Tricyclic antidepressants	Not detected		

NQ = not quantified

No alcohol measurements were undertaken on the samples from the later autopsy, and the alcohol results from the first autopsy were used for the interpretation.

Interpretation of Results

Carbon Monoxide and Cyanide

Death resulting from exposure to a fire may be due to a number of factors including direct physical trauma from the collapse of buildings, primary burns and secondary complications arising from them and the inhalation of toxic gases, vapours and particulates. The latter may produce local injury to the respiratory tract and/or toxicity following their absorption into the circulation. The toxic gases may also give rise to problems in judgement, impairment of co-ordination and alteration of consciousness level which can all hamper attempts to escape the fire danger.

It is common to have bodies discovered in places devoid of soot and remote from flames and heat, where death was clearly the result of inhaling the products of combustion. It has been estimated that up to half of the fatalities in fires are due to the inhalation of smoke and toxic gases.

A major constituent of smoke which has long been known to contribute to death is carbon monoxide (arising from the incomplete combustion of carbon containing compounds). Other toxic agents can also be produced, particularly where synthetic materials have been used in furniture and fittings. Synthetic materials, when burning, produce intense fires accompanied by the formation of copious amounts of smoke and toxic gases. Any material containing carbon and nitrogen, for instance (e.g. polyurethane), will liberate highly toxic hydrogen cyanide.

When air containing carbon monoxide is inhaled, the circulating blood is effectively offered a choice between the carbon monoxide and oxygen. Haemoglobin in the blood, which normally acts as the carrier for oxygen, has a very high affinity for carbon monoxide. This affinity is thought to be 200 to 300 times greater than that between haemoglobin and oxygen. The haemoglobin therefore combines with carbon monoxide, at the expense of oxygen, in a progressive fashion, until the blood eventually becomes saturated with carbon monoxide. The proportion of oxygen in the circulating blood falls below the level necessary to maintain the health of tissues notably the brain, heart muscle and the liver. This can lead to permanent tissue damage and death.

Symptoms of intoxication by carbon monoxide include headache, chest pain, confusion, visual disturbances and lassitude. Excessive exposure can lead to unconsciousness, respiratory failure and ultimately death. Carbon monoxide toxicity is increased by factors such as physical activity and cardiac disease. Death occurs when carboxyhaemoglobin saturation levels reach approximately 50% and above.

Cyanide also reduces the oxygen carrying capacity of the blood and inhibits enzymes responsible for cellular respiration, causing cell death. Therefore one would expect a combination of effects, to some degree, when both hydrogen cyanide and carbon monoxide are inhaled together. This is supported by data from previous cases where death has occurred from smoke inhalation in fires. In many of these instances the carboxyhaemoglobin saturation level was below 50% and the blood cyanide concentration was also below that which might be expected for death following exposure to cyanide alone (approximately 1mg/l and above).

In this instance carboxyhaemoglobin was detected at a low concentration in the liver serosanguinous sample; none was detected by Professor Kleiber in a serum

sample. This shows that Oury Jalloh had not inhaled a significant amount of carbon monoxide before he died but does not necessarily show that he was dead before the fire started. In some instances it is possible for a person to die very quickly from the effects of fire, through heat injuries or trauma, and in such instances they may inhale very little carbon monoxide. Indeed, if a fire burns in an open space there may actually be little carbon monoxide produced if there is sufficient oxygen to allow complete combustion. During this process carbon dioxide is produced rather than carbon monoxide. However, if Oury Jalloh had been exposed to smoke/fire fumes for more than a few minutes in a confined space, such as a cell, I would have expected carboxyhaemoglobin to have been detected, perhaps at a significant level but depending on the time of exposure.

When considering whether or not a person was alive when a fire started, toxicology results need to be considered with any other relevant evidence such as post-mortem findings, for example whether or not soot was present in the airways. This will be addressed by Dr. Walker in his report.

The low concentration of COHb detected in a sample taken at the later post-mortem examination is of a background concentration which would be expected to be present in smokers, however, the interpretation of COHb measurements on liver serosanguinous fluid should be treated with caution, because the matrix may not be the same as whole blood and could lead to measurement errors. The most useful evidential result is that for the post-mortem blood sample taken on the day of death which showed there to be no COHb present.

In a study by Yoshida et al. investigating 120 fire deaths, 9 were found to have a carboxyhaemoglobin concentration less than 10%.

A study by Yeoh and Braitberg examining fire deaths in Victoria, Australia looked at 178 victims and found that 52 had carboxyhaemoglobin concentrations less

than 10% of which 94% had what were considered to be "fatal burns". Indeed they further report that 43 of these victims had no carboxyhaemoglobin present.

With regard to sample stability on storage and delays prior to analysis, carboxyhaemoglobin has been reported as being relatively stable in preserved refrigerated blood specimens (Kunsman et al.).

Professor Kleiber did not detect COHb or cyanide in the serum sample taken at the first autopsy on the day of death. Studies have shown that the concentration of cyanide can fall significantly, especially between death and post-mortem sampling, although also, to a lesser degree, between sampling and analysis (Moriya & Hashimoto 2000 and 2001, Bright et al.). If samples are stored suitably, i.e. refrigerated or frozen, the decrease in cyanide concentration once the blood or serum sample has been taken should be small. Although it is not known what the limit of detections were for the two compounds clearly if either was present it could only have been at a very low concentration.

Cyanide was detected in Oury Jalloh's serosanguinous lung and liver fluid samples taken at the second autopsy. The concentration reported for the liver fluid was moderately high, that for the lung fluid much lower, however, these results should be treated with a degree of caution because there was a very long time between death and sampling for these samples.

There have been reports of cyanide being produced in samples after death, possibly by microbial action, although this should not occur if the sample is suitably preserved with fluoride (Lokan et al.). It is not known for any of the samples taken at the autopsies were suitably preserved with sodium fluoride and I cannot see any indication that fluoride was tested for by any of the Professors.

The absence of COHb and cyanide in the serum sample analysed by Professor Kleiber is likely to be the most relevant evidential result for this case, since the sample had been taken on the day of death. This result is entirely consistent with

the negative result for COHb and shows that Oury Jalloh had not ingested a significant amount of smoke/fire fumes before he died. The cyanide detected in the serosanguinous liver and lung fluid samples could have been produced in the body after death in the nearly 3 months before those particular samples were taken.

Ethanol (alcohol)

It is not known from what site in the body the post-mortem blood/serum samples were obtained, however, a pre-death sample was analysed as well as a post-mortem urine sample. All are in broad agreement once timings and distribution ratios are taken into consideration.

The blood alcohol concentration in the sample timed at 9.15am (2.98% = 2.98mg/g) would be expected to produce extreme drunkenness with effects such as significantly impaired consciousness, reduced reflexes, depressed respiration, incontinence and tending towards unconsciousness, absence of reflexes and even coma.

The blood alcohol concentration in the post-mortem sample was slightly lower, albeit still consistent with heavy drunkenness, and also consistent with the approximate concentration which might be expected allowing for nearly three hours of alcohol elimination between 9.15am and Oury Jalloh's death.

The detection of ethanol in the serosanguinous lung sample is entirely expected as ethanol will distribute into all of the water in the body and therefore will be present in any water-containing fluid, including serosanguinous fluid taken from the lung.

A table is included in Appendix 1 relating concentrations to expected effects in a social drinker.

Cocaine

Cocaine is rapidly metabolised in the body to a number of metabolites the major one of which is benzoylecgonine. Another metabolite is ecgoninemethylester (EME). A very similar compound is formed in the body when cocaine is taken when alcohol is present in the bloodstream, this compound being known as ecgonineethylester (EEE or cocaethylene).

Professor Kleiber reported cocaine to be present in the post-mortem urine sample. He also reported a concentration of <10ug/l in the serum. The latter result could be interpreted in two different ways. The result could mean that cocaine was detected but that it was present at a concentration less than 10ug/l (<10ng/ml) or alternatively it could mean that it was not detected at all and that the limit of detection for the method (the lowest detectable amount) was 10ug/l (10ng/ml); it is not known which of these is correct in this instance. Clarification could be obtained from Professor Kleiber.

He reported benzoylecgonine, the major body-breakdown product of cocaine, to be present at a concentration of 171.6ug/l, but also 142.7ng/ml in serum samples [please note that these units are interchangeable]. It is unclear which particular samples were analysed and when or where they were taken from. Benzoylecgonine was also detected in the post-mortem urine sample.

Professor Kleiber also reported detecting EME at concentrations of 54.8ug/l and 42.2ng/ml in the serum sample.

Professors Bratzke and Kauert reported detecting benzoylecgonine at a concentration of 0.17mg/l in the lung serosanguinous fluid and EEE at a concentration of 0.06mg/l in the same fluid. [Please note 1mg = 1000ug, therefore ug/l is equivalent to 1000 x mg/l and therefore 0.17mg/l is the same as 170ug/l and 170ng/ml].

I am not aware whether or not tests were performed for EME and cocaine as well on this sample.

The results of analysis of the serosanguinous lung sample cannot be interpreted reliably. The sample was taken nearly three months after death and is not a useful sample for quantitative analysis as there is no scientific data to use to help in understanding what the result means. It is adequate for screening purposes, that is to see whether a compound is present or not, but not for quantitative work.

Cocaine is very unstable and rapidly breaks down in the body after death and I would not expect it to be present in this lung serosanguinous fluid sample. Clarification from Professors Kleiber, Bratzke and Kauert would be needed to establish whether or not cocaine itself was detected. The reported concentration of EEE, assuming it was EEE rather than EME, again means nothing after this period of time as it too is unstable. It is unclear whether or not Professor Kleiber tested for EEE in his analysis.

As it is not certain at this stage whether or not cocaine was detected in Oury Jalloh's post-mortem serum sample the only reliable result we can use regarding unchanged cocaine is that from analysis of the post-mortem urine sample. Cocaine was detected in this urine sample which indicates use of cocaine within 12 hours or so of death.

Although it is unclear from which site the post-mortem blood/serum samples were taken there is nothing to suggest the use of cocaine in high dosage. The results are consistent with moderate to low abuse dosage within 12 hours or so of death. Due to the varied results reported it cannot be categorically stated whether or not alcohol was present in the bloodstream when the cocaine was

used. However, if the results do show that EEE was present, then it would indicate the cocaine was used when alcohol was present in the bloodstream.

Cocaine is a powerful stimulant drug which produces brief stimulant effects which then fade away with the comedown or after-effects then being experienced.

Following inhalation of smoke, the drug passes rapidly across the alveolar membranes in the lungs into the bloodstream, from where it is quickly carried to the brain. Smoking cocaine therefore results in rapid absorption, which leads to a rapid onset of effects, usually within a few seconds. This is colloquially referred to as a "rush" or "high".
If snorted the drug would be rapidly absorbed into the bloodstream via the nasal membranes with the effects commencing within a few minutes.

The initial stimulant effects are relatively brief and diminish after around 15 to 30 minutes or so and include an intense euphoria, feelings of increased energy, strength and self-confidence, exuberance, increased wakefulness and alertness, indifference to pain, talkativeness, increased heart rate and sweating. Cocaine will produce anaesthetic effects in localised areas (e.g. nose if snorted). Dilated pupils are produced.
The user will then experience after-effects ("come-down"), which can include depression, anxiety, fatigue, drowsiness, irritability, melancholy, disturbed sleep, hunger, lethargy, feelings of tension and agitation and craving for the drug. The duration of the after-effects is variable. This phase can also produce effects such as lack of concentration and co-ordination. Other drugs may be used to attempt to alleviate some of the comedown effects e.g. cannabis and benzodiazepine drugs.

A person who takes crack cocaine before retiring to bed would have great difficulty sleeping and users often take 24 hours or more after use before they can sleep properly.

The physical short-term effects of crack cocaine can include dry mouth, sore throat, sweating, loss of appetite, increased heart rate and raised blood pressure.

Cocaine users frequently attempt to recapture the initial "high", which can result in taking successive amounts in quick succession, colloquially referred to as "binge" usage. However, successive quantities taken over a short period will each produce less-intense euphoria than the preceding dose. Conversely, the greater the quantities used and the longer the duration of usage, the more severe the after-effects tend to be. If a person has used repeated quantities of the drug over a period of time, complete exhaustion eventually ensues. Cocaine binges may last 1 to 2 days with several days of recovery afterwards during which time no further cocaine is used.

Chronic, that is regular, heavy use of cocaine, can produce a number of effects. There is no reported clearly-defined physical addiction associated with 'crack' cocaine use, but it does produce psychological dependence. No physical illness generally results from cessation of use although there is often an intense craving for the drug.

Individuals who repeatedly use cocaine over a long period of time have an increased tendency to experience paranoia, anxiety, depression and psychosis.

Physical symptoms associated with regular usage of 'crack' include chronic coughing; wheezy breathing; and tightness in the chest. Chronic use of cocaine may cause damage to the heart and other organs, and increased blood pressure.

Regular users of cocaine powder may develop ulceration of the nasal passages, in turn leading to holes appearing in the nose in extreme instances.

Background information relating to cocaine is given in Appendix 2.

There have been many reports in the scientific literature, and I have dealt with others during my career, involving so-called 'excited delirium' (ED) following cocaine use. It is unclear whether or not this is applicable in this case but it is mentioned for completeness.

In instances involving ED the cocaine user will behave in a bizarre manner often attracting attention to themselves. Such cases frequently involve police being called to the person and having to restrain the individual. There have been many instances where force has been used, sometimes by police sitting on the individual, and requiring many officers to restrain the person due to the immense strength shown by some individuals. In some instances the individual has died whilst in police custody or whilst being restrained, with causation being due to extra demands being placed on the heart by a combination of cocaine use and restraint. In such instances the concentration of cocaine and/or benzoylecgonine in the bloodstream would be expected to be high if the samples are taken relatively quickly.

In this instance there was no suggestion of either cocaine or benzoylecgonine having been present at a high concentration although cocaine can breakdown if a sample is not preserved correctly. There is no indication in this case of whether or not the analysed post-mortem blood sample was suitably preserved. Analysis for cocaine and benzoylecgonine on the sample taken prior to death would have assisted also.

In this instance it appears that Oury Jalloh drew attention to himself by his behaviour and police were called. It is not known what attempts were made to restrain him but clearly he was cuffed in his cell due to his behaviour.

Given the state of his body after the fire I am not able to comment on what evidence might remain to show whether Oury Jalloh had suffered any cardiac consequences from cocaine use and police intervention.

Cannabis

The results are consistent with Oury Jalloh having used cannabis or cannabis resin previously.

No analysis for THC appears to have been performed or, if it was, then no result was reported. Only a result for THC-acid has been reported. As with the cocaine result the THC-acid result for the serum sample reported by Professor Kleiber could be interpreted in two different ways. The result could mean that THC-acid was detected but that it was present at a concentration less than 10ug/l (<10ng/ml) or alternatively it could mean that it was not detected at all and that the limit of detection for the method (the lowest detectable amount) was 10ug/l (10ng/ml); it is not known which of these is correct in this instance. Clarification could be sought from Professor Kleiber.

No testing for cannabis use was reported by Professors Bratzke and Kauert.

A significant THC concentration would be consistent with recent use, but in the absence of any test for THC it is not possible to estimate the time of last cannabis use by Oury Jalloh. There it is not possible to say whether or not he would have been under the influence of cannabis at the time of his arrest or death.

This is because it is well known that THC-acid, the major body-breakdown product of THC, can persist in the body for a long time after last use, particularly in heavy, regular users of the drug.

Therefore although the result of analysis of the urine sample, and possibly also the serum sample, shows prior use of cannabis it is not possible to say from these analytical results when, since the results obtained could have arisen from last use several days previously.

The concentration of THC-acid detected in the urine sample is very low; this would suggest non-recent use of the drug. A small amount of THC-acid can be present in a urine specimen following passive inhalation, that is the ingestion of smoke from another person's cannabis cigarette, and the low concentration of THC-acid detected could theoretically have arisen from recent ingestion of another person's cannabis smoke. It should be pointed out however that it would appear from information supplied that Oury Jalloh could not have been exposed to cannabis smoke for 3 hours before he died as was in police custody for that period of time. Therefore the THC-acid detected in his urine specimen would have been present from prior use of cannabis or cannabis resin rather than via passive inhalation. Taking all of the results into consideration there is no evidence to suggest that Oury Jalloh had used cannabis recently although use of a small amount recently cannot be discounted.

A recent paper (Fabritius et al.) has investigated the use of <u>THC-acid</u> concentrations as a discriminator of occasional versus heavy cannabis use. They concluded that a blood THC-acid concentration of less than 3ng/ml, which would equate to a serum concentration of less than 6ng/ml, would be consistent with occasional use whereas blood THC-acid concentrations in excess of 40ng/ml would suggest heavy use, which they defined as 10 or more joints per month. In this case it is unclear whether THC-acid was actually detected in the serum sample but, if it was, then it was less than 10ug/l (<10ng/ml) which shows that Oury Jalloh was not a heavy user of cannabis.

Further information regarding the use and effects of cannabis is given in Appendix 3.

Drug and Alcohol Interactions

There is evidence that Oury Jalloh used cocaine relatively recently prior to arrest and death and had also consumed a large amount of alcohol. He had used cannabis although it is not possible to say when. Any doubt on whether or not EEE was present is because of possible errors in transcription/translation of the reports, and not because of any suggested error in the analysis.

If cocaine is used when alcohol is present in the bloodstream an interaction, which has been well studied, can occur. Another compound, known as cocaethylene, is formed in the body. The outcome of this is that the overall stimulant effects will be prolonged (possibly doubled) and the comedown is reported to be less intense. In this case it is unclear whether or not cocaethylene (ecgonineethylester, EEE) was detected. The toxic effects of cocaine, especially those on the heart, are reported to be more enhanced if used in combination with alcohol due to the formation of cocaethylene (Awtry et al., Pennings et al.).

Cannabis is sometimes used to alleviate some of the comedown effects produced by stimulant drugs such as cocaine. In this instance it is unclear whether this interaction may be of relevance as there was nothing to suggest recent use of cannabis by Oury Jalloh, although recent use of a small amount cannot be discounted.

Conclusions

1. The best evidential analysis for carboxyhaemoglobin (COHb) was that performed on the post-mortem blood sample taken on the day of death, which was negative. This result shows that Oury Jalloh had not inhaled a significant amount of smoke/fire fumes before he died. The absence of

cyanide reported in sample(s) taken at the post-mortem examination is also consistent with this.

2. Oury Jalloh had used cocaine relatively recently, within 12 hours or so, before his death.
3. He had also consumed a large amount of alcohol and his blood alcohol concentration would be consistent with heavy drunkenness in a normal social drinker.
4. Use of cocaine and alcohol concurrently will lead to a prolongation of the stimulant effects of cocaine and in the possible toxic effects which can be produced by cocaine use.
5. Oury Jalloh had attracted attention by his behaviour. His use of cocaine, together with forcible restraint by police, could have produced so-called excited delirium. There have been a number of reported deaths following such a scenario although it is not known from the toxicology results whether this is relevant in this case.
6. Oury Jalloh had also used cannabis previously although there is no evidence to suggest recent use of a large amount of the drug. Recent use of a small amount cannot be precluded and the results are consistent with Oury Jalloh having been an occasional user, rather than a heavy regular user, of cannabis.

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Appendix 1 -Alcohol

Alcohol effects by blood concentration

The effects detailed below are for guidance only and may not apply to any specific individual.

Although categorised for ease of reference, the symptoms can overlap considerably and gradually increase in severity with increasing concentrations. Whilst the effects described could apply to a social drinker, they depend on the person's degree of habituation. A heavy drinker would be expected to show less-noticeable effects and a person unaccustomed to alcohol more pronounced symptoms. Therefore the effects of any given blood alcohol level cannot be obtained simply from the table. Tolerance should be considered, together with other information such as evidence from other witnesses and the findings of any medical/psychiatric examination.

0.09 – 0.47mg/g	Little outward effects, feelings of relaxation and well-being, increased sociability
0.47 – 0.96mg/g	Increased self-confidence and talkativeness, mild euphoria, reduced co-ordination and slightly slowed reactions.
0.96 – 1.44mg/g	Impaired balance, thickened speech, clumsiness, reduced alertness, lowered social reserve, increased garrulousness and volubility
1.44 – 1.92mg/g	Drunkenness, slurred speech, glazed eyes, flushed complexion, staggered gait, drowsiness, exaggerated emotional responses, impaired co-ordination, reduced inhibitions, dizziness, nausea, disorientation
1.92 – 2.4mg/g	Marked or heavy drunkenness, confusion, grossly impaired co-ordination, vomiting, reduced awareness, short-term

	memory may be impaired
2.4 – 2.88mg/g	Extreme drunkenness, stupor, impaired consciousness, reduced reflexes, depressed respiration, incontinence
2.88 – 3.84mg/g	Unconsciousness, absence of reflexes, coma
>3.84mg/g	Possible death by respiratory depression or cardiac arrest.

Appendix 2 -Cocaine

Background information

What is cocaine?

Cocaine is a readily available drug in the UK and is very frequently encountered. It is encountered in two forms, either as cocaine hydrochloride, a white or cream-coloured glistening powder commonly known as snow, blow, white lady, or in the form of waxy-looking cream-coloured lumps commonly referred to as crack, rock or freebase.

The powder form can be readily made into 'crack' with a little basic knowledge and other materials. 'Crack' cocaine is sometimes of high purity (60-80% or so) but in the last couple of years the purity has dropped significantly. Formerly cocaine hydrochloride powders were often in excess of 70% pure as well but many are now less than 20% pure.

Crack cocaine is produced by heating cocaine hydrochloride powder with an aqueous alkali such as a solution of sodium bicarbonate or ammonia in water. This converts the cocaine to the base form, which is insoluble in water and separates out as a yellow oil. This cools to form an off-white solid. This is then broken into off-white lumps ready for use.

Why and how is it used?

Cocaine is a stimulant drug abused for its reported effects of producing an excited state of euphoria and is normally taken either by:-

i) nasal insufflation ("snorting") of the powder through a tube or

ii) smoking "crack" in either a cigarette or via a home-made pipe.

A typical abuse dose of cocaine used to be of the order of 100 to 200 milligrams of powder when the powder strength was higher than currently encountered

and therefore nowadays a typical abuse dose of illicit cocaine powder is probably higher than this.

When 'binge' use of cocaine occurs, however (see below for details), a much larger amount of drug would be used. This could be up to perhaps a gram in moderate 'binge' use or up to 5 grams in heavy 'binge' use, perhaps over a period of 1-2 days or so. 'Binge' use of cocaine, however, would not be regarded as normal use of cocaine.

Appendix 3 -Cannabis

Background Information

What is cannabis?

Cannabis is a plant (*Cannabis sativa*), also known as hemp, which is harvested for its drug content. Cannabis is a Class B controlled drug under the Misuse of Drugs Act 1971 and is the most widely used illegal drug in the United Kingdom. Forms of cannabis include herbal material, resin and oil. Cannabis resin is a concentrated form of the herbal material with the parts containing the greatest concentration of active component being preferentially removed and formed into hard blocks; cannabis oil (hash oil) is a liquid extraction of cannabis evaporated down to further increase the concentration of the active component.

Why and how is it used?

It is a mild sedative which is abused for its euphoric and relaxing properties. It is commonly known as hash, marijuana and ganja with the more potent recent forms being frequently referred to as skunk or sinsemilla.

The drug is normally used by smoking it in the form of a cigarette commonly referred to as a 'reefer', 'joint' or 'spliff'. However, the drug can also be smoked via a pipe, sometimes of an elaborate construction called a 'bong', and is occasionally taken orally in the form of cakes or other confectionery.

The United Kingdom's House of Lords Select Committee on Science and Technology Ninth Report contains definitions of recreational cannabis users, namely casual users, regular users and heavy users.

Casual users are defined as irregular users smoking cannabis in amounts of up to 1 gram at a time but not more than 28 grams per year.

Regular use is defined as typically ½ gram per day in 3-4 joints (i.e. about 150mg cannabis per cigarette) adding up to about 3½ grams per week.

Heavy use is defined as more than 3½ grams per day and 28 grams or more per week and describes the group as being "more or less permanently stoned".

What general effects does cannabis produce?

1. Short-term

The main active constituent of cannabis and cannabis resin is Δ^9-tetrahydrocannabinol, also known as THC, which is converted to 11-hydroxy-THC (also known as THC-OH). These compounds are both pharmacologically active but are quickly eliminated from the bloodstream, being converted to the inactive metabolite 11-nor-Δ^9-tetrahydrocannabinol-9-carboxylic acid (also known as carboxy-THC or THC-acid).

There are many other cannabinoids present within the plant material, including cannabidiol (CBD) and cannabinol (CBN). In the last 10 years or so very potent strains of cannabis have been developed and which are now frequently grown in the UK in roof spaces, spare rooms and even on an industrial scale in warehouses. Such strains of cannabis may contain THC at a concentration in excess of 25% when harvested, although the normal value may be closer to around 10%; however this is still much stronger than foreign imported cannabis which normally has a THC strength of around 3 to 4%.

Cannabis is normally used by smoking, when the effects begin within seconds, peak between 15 and 30 minutes and last for 2 hours or so, but perhaps occasionally up to 4 hours, following use of a 'normal' single abuse amount. However the duration of effects will depend on factors such as the quantity and potency of the material used, and the number and depth of inhalations. A typical

dose of THC by smoking would be approximately 5 to 20 milligrams (mg). The onset of effects is slower and the effects less intense if the drug is taken orally.

The effects of cannabis are reported to depend on the amount used, the social setting, the user's expectations and previous experience. The main effects are euphoria, relaxation, drowsiness, reduced alertness, distortion of the perception of space and time, impairment of short-term memory and judgement, and some loss of co-ordination. If used in company, the drug can increase sociability with a tendency for talkativeness and hilarity. A common side-effect of the drug seen in persons who are under its influence is reddening of the whites of the eyes ('bloodshot eyes'). The user's heart rate will increase and dry mouth and throat are common. A more vivid sense of smell, taste, sight and hearing is regularly reported. Hunger pains, referred to as 'the munchies', are common. Persons unaccustomed to the drug may experience nausea or vomiting.

More potent varieties of herbal cannabis are known as 'skunk' and are becoming increasingly common. The effects from these varieties are likely to be more intense and may last for longer than non-skunk varieties. Higher doses can produce anxiety, disorientation, confusion, restlessness and panic reactions.

2. Long-term

Long-term heavy regular users of cannabis may be more prone to develop paranoia and psychoses, and regular use can lead to tolerance and dependence. Prolonged heavy consumption can lead to apathy, lethargy and impaired social functioning. Withdrawal symptoms following chronic usage can include insomnia, irritability, anxiety and depression.