“Long Term exposure to Carbon Monoxide: National Study”

Final Report

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Executive Summary

In September 2011, the department of health published statistics indicating that over 4000 people in England and Wales were diagnosed with CO poisoning and in the same month, the Gas Safety Trust published figures showing that there were 85 deaths in 2009 due to accidental exposure to CO. The department of health reports highlight that this figure is probably under represented as the symptoms are not identified and are assumed to be linked to other illnesses. This research aims to explore the levels of understanding of the issues around Indoor Air Quality (IAQ) and Carbon Monoxide (CO) poisoning of the public in the United Kingdom, as well as monitoring the continuous exposure to the low levels of CO. A research project was set up with Merseyside Fire and Rescue, West Midlands Fire and Rescue, Coventry Council and Liverpool John Moores University. The first phase of this project was to utilize the Home Fire Safety Check (HFSC), which the fire services had already conducted and extend this to include CO specific questions, and instantaneous CO reading within the house, taken while the fire personal are in the property. The second phase of the project was to place CO data loggers within the properties, and measure CO levels for 2 weeks to see if any trends could be identified. The outcome from the first phase of the project include that, only 10% of households in Liverpool, and 3% of households in Coventry had a CO alarm even though 80% of these households have gas heating or cooking. The outcome from the second phase was that in all of the 174 properties (selected by random) there were levels of detectable CO and 25% of these included spikes of relatively high levels that are a concern for potential health risks and an indicate that an appliance may be faulty and need servicing. The strength of these results shows the potential risks of both long-term CO exposure and the public's CO awareness.

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1. Introduction

1.1. Background

Carbon monoxide (CO) is a highly poisonous colourless and odourless substance that results from the incomplete combustion of a fuel source (Tam *et al.*, 2012). The main causes of CO in homes are when gas appliances have been poorly fitted, maintained or repaired, but leaks may also occur if vents, flues and chimneys are blocked, and come in through adjoining properties. According to the Department of Health (Davies, 2010), approximately 50 people lose their lives from acute carbon monoxide poisoning in England and Wales, and in excess of 200 people require hospital admission as a consequence of non-fatal carbon monoxide poisoning, every year. It has long been believed that many cases of carbon monoxide poisoning may be unrecognised and/or misdiagnosed (Walker and Hay, 1999), as many symptoms mirror common conditions such as influenza or food poisoning (Tam *et al.*, 2012). As the cooler seasons result in a proliferation of viral type illnesses as well as the likelihood of exposure to CO, it has been suggested that misdiagnoses may occur (Ernst and Zibrak, 1998).

Those people who seek medical help due to CO may experience negative and long-term after-effects because of their poisoning. This can result from lower-dose or chronic exposure as well as a higher-dose and significantly higher-risk experience.

Physiological effects include a wide range of diverse symptoms:

- Headache, dizziness, seizures, dyspnoea, chest pain, myocardial ischaemia, ventricular arrhythmias, pulmonary oedema, nausea, and vomiting (Chun *et al.*, 2012). Myocardial depression, hypotension and arrhythmia may also present (Prockop and Chichkova, 2007), along with syncope, incontinence, seizures, coma and death (Roderique *et al.*, 2015).

Neuropsychiatric symptoms include:

- Memory and executive function impairment, apraxia, aphasia, and agnosia, slow mental processing speed and reduced intellectual functions, lethargy, Parkinsonism, hallucinations and motor dysfunction (Prockop and Chichkova, 2007)(Roderique *et al.*, 2015)

Arguably, the most serious neurological condition caused by CO poisoning is a neuropsychiatric syndrome, which can present during the initial phase or over many subsequent weeks. This is caused by lesions in the brain, most notably in the basal ganglia/globus pallidus region (Katirci and Kandis, 2010), leading to a collection of conditions that may include:

- Emotional disorders and the decline of cognitive function, dizziness, paraesthesia, lethargy, somnolence, motor and sensory disorders, dementia, personality and judgement disorders, encephalopathy and neuropathy (Prockop and Chichkova, 2007); Parkinsonism, dystonia and acquired Obsessive Compulsive Disorder (OCD)

The HPA (Health Protection Agency, 2011) add that these symptoms are more common in those over 40 years of age. The authors also include gait disturbance and urinary/faecal incontinence, citing parietal lobe lesions as a factor.

Lee, Li and Ao (Lee, Li and Ao, 2002) found that homes which rely upon gas appliances for cooking, or homes with smokers had lower quality of indoor air than homes with no smokers or gas appliances (Jones *et al.*, 2000). As the majority of properties in and around Liverpool rely on gas as their main source of heating and cooking, a study to assess the level of awareness that residents have of CO is necessary.

In 2010, Liverpool John Moores University (LJMU) embarked on a project looking into the presence and identification of Carbon Monoxide (CO) in premises within Liverpool and Coventry supported by Merseyside Fire and Rescue Service (MFRS) and West Midlands Fire and Rescue Service (WMFRS).

The initial reading within the property was taken using a ToxiRAE single gas detector and in addition to the standard home safety questionnaire with some additional questions regarding CO.

During the period of the study, Merseyside recognized the value of the ToxiRAE detectors, and issued them to all front line appliances and staff.

The study resulted in around 32,000 sets of data being captured. For academic credibility, this data need to elaborate and reduce to 22,000 data sets. The standout result of this data was that out of 22,000, only 49 readings were recorded over 0ppm.

This prompted a new approach, and led to the second phase, which introduced the use of Lascar Data Loggers. These were programmed to take samples at 1 minute intervals for a period of 2 or more weeks as advised by Public Health England. The addresses targeted were those that had previously been visited during Phase 1 and recorded a zero reading. The results were startling as a large numbers of these properties actually had levels of CO that were above the World Health Organisations long term exposure (LTE) level of 9.6ppm.

Commented [KP1]: How many?

This piece of work demonstrated that low level long term exposure risks did exist, so in 2013, LJMU and MFRS started a larger piece of work across the UK supported by 8 other FRS's to gain additional data to support the initial findings.

1.2. Aim and Objectives

The aim of this research project was to investigate the long-term exposure to low level of Carbon Monoxide across the United Kingdom.

To achieve the research aim the following objectives were established:

- Investigate the indoor distribution and effect of long term exposure to low levels of Carbon Monoxide
- Distribute Carbon Monoxide sensors for a national study
- Collect experimental data using data loggers and interviews with people that had experienced exposure to Carbon Monoxide
- Conduct data analysis to understand the public awareness with regards to exposure to Carbon Monoxide

2. Current State of the Art

2.1. Current Practices and Guidance

According to Professor Dame Sally Davies *et al* (Davis, Cummings and Bennett, 2013) carbon monoxide poisoning can be divided into two different categories namely accidental (and therefore preventable), which still result in recorded cases of around 40 deaths and 200 hospitalisations each year in England & Wales. Recent figures from the

Department of Health indicate that there are 4,000 attendances at accident and emergency departments for treatment for CO poisoning each year in England. While a considerable number of people die from accidental acute carbon monoxide poisoning, it is now confirmed many more are injured by sub-lethal exposure. The second category covers carbon monoxide poisoning, especially when there has been long-term exposure to low concentrations, is notoriously difficult to diagnose because the symptoms often mimic other more common illnesses such as flu and food poisoning. The most common symptom of carbon monoxide poisoning is headache, Misdiagnosis leading to treatment of only the symptoms of poisoning my lead to the patient being sent home where exposure might continue, leading to serious, perhaps fatal consequences. Sub-lethal levels of carbon monoxide poisoning can lead to chronic health problems. Public Health England (Public Health England, 2016) reports that the Carbon monoxide is produced when fossil fuels burn without enough oxygen. One of the most important source of exposure to carbon

monoxide for the general public is from cooking or other fuel burning appliances which are poorly installed, faulty or used inappropriately (including inadequate ventilation). For example, home boilers that are installed incorrectly. The use of BBQs and portable generators inside homes, caravans and tents. Inhaling smoke from a house fire may lead to carbon monoxide exposure. For smokers, cigarettes are the major source of carbon monoxide. Exposure to low levels of carbon monoxide can occur outdoors as it is produced by vehicle exhausts and industrial processes.

When inhaled carbon monoxide enters the blood through the lungs and attaches to the body's oxygen carrier, haemoglobin. This reduces the amount of oxygen that can be carried round the body. A brief exposure to small amounts of carbon monoxide may cause headache, flushing, nausea, dizziness, vertigo, muscle pain or personality changes. Exposure to higher amounts may cause movement problems, weakness, confusion, lung and heart problems, loss of consciousness and death.

The current guidance from Public Health England in the situation that you think you can be exposed to carbon monoxide is:

> *If your carbon monoxide alarm sounds or you believe there to be a leak (unignited gas does not contain carbon monoxide):*
>
> *Stop using all appliances and evacuate the property immediately (try to stay calm and avoid raising your heart rate)*
>
> *Call the gas emergency number on 0800 111 999 to report the incident or the Health and Safety Executive (HSE) Gas safety Advice Line on 0800300363*
>
> *Do not go back into the property (wait for advice from the emergency services)*
>
> *Get immediate medical help*
>
> *If you think you have been exposed to carbon monoxide and feel unwell or are worried call NHS 111, contact your GP or in an emergency contact emergency services 999. When people are removed from the source of carbon monoxide exposure, their health usually improves and their symptoms subside, but it is still important to seek medical advice.*

While it is considered extremely hazardous to endure any period to an atmosphere that contains more than 100 parts per million (ppm) of CO (Chavouzis and Pneumatikos, 2014), the World Health Organisation (WHO 1999/2004), (World Health Organization, 2010) recommends that we should not be exposed to more than 87ppm for 15 minutes as a safe limit. Public Health England recommend the same guidelines, with occupational standards of 30ppm for an 8-hour reference period and 200ppm for a 15-minute reference period (Public Health England, 2016):

***Table 1**. Public Health Guidelines*

Indoor air quality guideline	100 mg/m 3 (87 ppm) for 15 minutes 35 mg/m 3 (30 ppm) for 1 hour 10 mg/m 3 (8.7 ppm) for 8 hours 7 mg/m3 (6.1 ppm) for 24 hours

The United States Environmental Protection Agency use slightly different indicators as standards, with 9ppm as an 8-hour average and 35 ppm as a 1-hour average. These standards have not been changed since 1971, when they were established to provide the necessary protection for the public while including an 'adequate margin of safety' (United States Environmental Protection Agency, 2011). Although death and injury from CO poisoning has fallen since the time when these standards were established, people continue to lose their lives, their loved ones and their health from CO poisoning. The survivors may have to contend with distressing symptoms, with or without a formal diagnosis (Walker and Hay, 1999), or without support (All-Party Parliamentary Carbon Monoxide Group, 2015).

While this section seeks to explore some of the issues that affect the public's safety with regard to this gas, and highlight some of the areas where we do not have clear answers, Table 2 sets out a summary of some of the

common perceptions of CO. The persistence of these ideas contribute to continued death and injury from CO. Despite various educational and behaviour change campaigns, people's ability to protect themselves from this preventable danger, through knowledge and awareness, remains limited; the rate of admissions has both decreased and increased again across the years 2001-2010 (Ghosh *et al.*, 2015). The diagnosis and treatment of carbon monoxide poisoning may also be inadequate at times. These ideas form a template for this section, as each of the points will be addressed in turn.

***Table 2**. Commonly held perceptions about CO.*

Perceptions	
Acute and chronic poisoning	Acute poisoning – a dose that is large enough to cause injury/death quickly, usually occurring over a relatively short period. Chronic poisoning – an accumulative poisoning whereby a lower, non-fatal dosage occurs, and may persist, causing enduring yet seemingly non-specific symptoms.
If it's not fatal, you're fine	The perception is that recovery from CO poisoning is relatively straightforward and invariably complete. Both acute and chronic forms of CO poisoning, however, lead to diverse and lingering symptoms
Oxygen starvation of tissues (hypoxia) is the main issue	Although significant, hypoxia and the formation of Carboxyhaemoglobin are only a part of the situation; CO is a toxin
A certain level of exposure/poisoning will correspond to certain symptoms	Symptoms are varied and the issues are complex. It is known that different individuals present different symptoms

2.1.1. Acute and Chronic Poisoning

There is an abundance of literature on CO and its effects. Much of this is case study reporting, which tends to explore acute episodes of high-dose CO poisoning, and the consequences on various organ systems of the survivors (Greingor *et al.*, 2001; Liang, Chou and Yang, 2011; Abdulaziz *et al.*, 2012). Sykes *et all* (Sykes and Walker, 2015) describes the 'not uncommon' circumstance for healthcare professionals of a near-fatal incident of exposure brought to the awareness of the emergency services, which reflects the literature on CO.

Far fewer examples discussing lower level or chronic poisoning exist, and the survivors of these incidents remain unheard. As rightly pointed out by (Bolton, 2016), this is due at least in part to the difference between the definitions of the terms 'exposure' (where there is the potential for contact with a toxic substance, but without necessarily incurring immediately noticeable adverse health effects) and 'poisoning' (where there are adverse health effects resulting from inhalation of CO). The way these terms are used in the literature, where low-level poisonings may be excluded and any symptoms being regarded as not related to CO, has a significant impact on our understanding of the subject area, and more work needs to be done to address this issue. (Wright, 2002), defines poisoning as acute, chronic, and occult. Acute poisoning presents as serious symptoms often following a single exposure to a large amount of CO, involving one or several people who have been affected by the same, identifiable source. Acute poisoning has also been described as exposure for up to 24hrs, while chronic poisoning is exposure for longer than 24hrs, including intermittent exposure (Sykes and Walker, 2015).

Chronic poisoning represents a large percentage of poisonings, but despite it having a significant impact on individuals (Bolton, 2016), far less focus is given to its discussion in the literature. It may be indicated after exposure of more than one occasion to CO, usually at far lower concentrations, when the person or persons affected find themselves to be unwell. Repeated, long-term exposure and the resulting symptoms may prompt those affected to seek help from healthcare professionals. Occult (hidden) poisoning may never come to the attention of a healthcare professional, as if it is a result of chronic poisoning, as it often is, the affected individuals themselves may not seek

help. Acute poisoning may also be classed as occult if misdiagnosis by a healthcare professional occurs (Wright, 2002).

(Wright, 2002) describes CO poisoning as a pyramid of presentation conditions, where the apex is acute poisoning and the base is occult, low level exposure. The extent of the base of this pyramid remains poorly understood.

It has long been believed that many cases of CO poisoning may be unrecognised and/or misdiagnosed (Walker and Hay, 1999), as continued exposure to lower CO levels results in many non-specific symptoms that mirror common conditions such as influenza or food poisoning (Tam *et al.*, 2012), or which make diagnosis difficult (De Juniac *et al.*, 2012). Some of these symptoms may be relatively minor, but some are very difficult to live with. As the cooler seasons result in a proliferation of viral-type illnesses, as well as the increased likelihood of exposure to CO, this too can contribute to misdiagnoses (Ernst and Zibrak, 1998).

Acute poisoning is known to be non-specific and highly variable in its presentation (Wolf *et al.*, 2017). The central nervous system is most sensitive to CO, leading to headache, dizziness or dysfunction (memory and attention issues, lethargy and fatigue) and progressing to syncope, seizures and coma (Whitson, Lim and Poonai, 2011), hypotension, severe acidaemia, and respiratory collapse (Wolf *et al.*, 2017). We can add vomiting and diarrhoea, confusion, angina and breathlessness (Wright, 2002); mood disturbance, personality changes and dementia (Yeh *et al.*, 2014).

Chronic CO poisoning leads to similar, non-specific issues; flu-like feelings (although the affected people remain apyrexial) are common, as are symptoms that are similar to food poisoning. Symptoms usually progress as the source of the poisoning remains difficult to identify and other conditions are investigated and discounted (Seale, Ahanger and Hari, 2018). Table 3 shows some of the symptoms of both conditions.

Table 3. Symptoms of carbon monoxide poisoning (Public Health England, 2016; Seale, Ahanger and Hari, 2018)

Acute poisoning	**Chronic poisoning**
Headache	Impaired concentration
Nausea, vomiting and diarrhoea	Headache
Vertigo/dizziness	Lethargy and fatigue
Alteration in consciousness	Syncope (fainting)
Subjective weakness	Nausea
Confusion	Flu-like symptoms
Myocardial infarction/angina/chest pain	Anxiety
Respiratory failure	Psychomotor dysfunction
Loss of consciousness	Loss of balance
Seizures	Changes in sleep, memory, vision
Death	Hearing loss
	Emotional lability and impulsive behaviour

2.1.2. "If you don't die, you're fine"

Despite the lack of understanding regarding the long term effects and problems that are caused by either acute or chronic CO poisoning, it is clear from the literature that those who are exposed to CO may experience these negative after-effects as a consequence of any kind of poisoning. Although many may think that after oxygen therapy, if that is needed, anyone who is affected will recover, this is not the case. Indeed, CO toxicity may increase the brain's "constitutional vulnerability" to psychiatric symptoms (Chun *et al.*, 2012). CO poisoning has also been said to be characterised by a delayed onset of significant symptoms (Kondo *et al.*, 2007). Whilst some of these symptoms may be relatively minor, albeit difficult to live with, they occur because poisoning can have lasting and significant neurological sequelae as an outcome (Davies, 2010). The onset of neuropsychiatric changes that lead to these sequelae vary from one area of the brain to another, which may have some bearing on the fact that those who are poisoned may experience them immediately, or there may be a delay in their development which can be a significant period of time (Chun *et al.*, 2012). (Beppu, 2014) postulate that many who have experienced acute CO poisoning and then present with neuropsychiatric symptoms will see those symptoms resolve following treatment with oxygen.

Of the remainder of this population, estimated at 30% of those affected, approximately two thirds will experience persistent symptoms, and the rest will develop a Delayed Neuropsychiatric Syndrome (DNS). This is described as appearing over many subsequent weeks (Chun *et al.*, 2012), or between 2-40 days (Beppu *et al.*, 2011) or 3-240 days after exposure (Chavouzis and Pneumatikos, 2014). Symptoms can recur after periods of seeming recovery (Kao and Nanagas, 2006), after demyelination has advanced beyond "some unknown threshold" (Beppu *et al.*, 2011).

Neurological symptoms include memory and executive function impairment, apraxia, aphasia, and agnosia, slow mental processing speed and reduced intellectual functions, lethargy, Parkinsonism, hallucinations and motor dysfunction (Prockop and Chichkova, 2007). A characteristic feature of this sort of delayed encephalopathy are periventricular white matter lesions (Kondo *et al.*, 2007). MRI scans can be useful in identifying lesions in affected persons, which most notably occur in the basal ganglia/globus pallidus regions, the hippocampus, the deep white matter, and frontal lobe (Yeh *et al.*, 2014) and parietal lobe (Chavouzis and Pneumatikos, 2014). Indication of such damage is manifest in a collection of conditions that may include emotional disorders and the decline of cognitive function, dizziness, paraesthesia, lethargy, somnolence, motor and sensory disorders, dementia, personality and judgement disorders, encephalopathy and neuropathy (Prockop and Chichkova, 2007). In addition, as previously mentioned, pseudo-Parkinsonism also occurs, as can dystonia and acquired Obsessive Compulsive Disorder (OCD) (Katirci and Kandis, 2010). Depression, diurnal bruxism and psychic akinesia (Liang, Chou and Yang, 2011) may also follow, as may gait disturbance and urinary/faecal incontinence (Chavouzis and Pneumatikos, 2014).

Recovery from DNS or indeed regression of any of these symptoms is variable, but prognosis may indicate 50-70% remission of symptoms in a year (Chavouzis and Pneumatikos, 2014). (Pepe *et al.*, 2011) state that resolution of symptoms can occur over months but may be permanent in around 25% of cases.

2.1.3. Oxygen starvation of tissues (hypoxia) is the main issue

A reason for the lack of knowledge among the general public and some healthcare professionals about the sequelae of CO poisoning is perhaps linked to the idea that hypoxia is the most significant aspect of CO poisoning, or, indeed, the only mechanism that is mentioned by some authorities. Although hypoxia is a critical and very damaging element of poisoning, this assumption may mean that the natural, logical idea would be that once CO is no longer being inhaled, harm stops occurring to the body and brain; once the individual starts to recover from the hypoxia, recovery will commence from that point. This preconception not only potentially underestimates the effects of hypoxia, it does not consider the other mechanisms whereby CO poisons people, which will be explored in this section.

Cellular hypoxia is certainly a considerable factor in CO poisoning and, as mentioned, causes substantial damage to those who are poisoned. The tissues of the body are starved of oxygen during poisoning, due to the occupation of the inhaled CO molecule on the oxygen binding sites on the haemoglobin molecule, for which it has a high affinity. This molecule is known to bind preferentially with haemoglobin; it has 240-250 times more binding power with haemoglobin than the oxygen molecule does (Abdulaziz *et al.*, 2012) (De Juniac *et al.*, 2012). This binding reaction creates carboxyhaemoglobin (COHb or CO-Hgb) (Roderique *et al.*, 2015). COHb thus inhibits the amount of oxygen carried in the blood and impedes oxygenation of cellular tissue (Kokkarinen *et al.*, 2014), leading to a relative anaemia (Haldane, 1972; Kao and Nanagas, 2006) and symptoms such as dizziness, breathlessness, fatigue and headache. The presence of CO also induces an oxygen dissociation curve shift to the left, thereby decreasing the ability of any remaining (unaltered) haemoglobin molecules to release oxygen to the tissues (Chavouzis and Pneumatikos, 2014). The oxygen-carrying capacity of the blood is therefore compromised (WHO 1999/2004) and the individual is essentially in danger of suffocation. Crucially, the resultant COHb molecule also impairs the release of oxygen from oxyhaemoglobin (Environmental Protection Agency, 2010). Oxygen is within the body, but cannot be released to go where it is needed. Under such circumstances, certain compensatory alterations in haemodynamic responses will attempt to ameliorate the effects of hypoxia; these include vasodilation and increased cardiac output, which would benefit otherwise healthy individuals who were breathing ambient air, but may well exacerbate the deleterious effects of the CO poisoning. Once removed from the source of the CO, however, binding of CO to the haemoglobin molecule reverses and oxyhaemoglobin can be formed again.

The assumption that the damage occurs through the toxic mechanism of hypoxia secondary to hypoxaemia does not, by itself, explain much of the currently available clinical data (Gorman *et al.*, 2003); in particular, the delayed effects of the damage that CO causes. It appears that the way that CO acts as a is multifactorial poison (Wolf *et al*., 2017). Research remains ongoing into other mechanisms of poisoning and of CO's role as a gasotransmitter (Bleecker, 2015; Roderique *et al.*, 2015)

It is suggested that the clinical effects of CO poisoning perhaps result from a combination of hypoxia/ischaemia due to COHb formation and direct CO toxicity at a cellular level (Kao and Nanagas, 2006), provoking both an immunological and inflammatory response (Chavouzis and Pneumatikos, 2014); leading to demyelination of the Central Nervous System (CNS) (Beppu *et al.*, 2011). There is also a direct mitochondrial electron transfer dysfunction (Abdulaziz *et al.*, 2012); essentially, a poisoning of cells. Further damage to tissues also takes place due to reoxygenation/post-ischaemic reperfusion injury (Ernst and Zibrak, 1998). The combination of these effects results in a hypoxia-driven succession of complications, some short-lived and some significantly longer-term.

CO also readily binds to several other proteins which contain haem (and are distinct from haemoglobin and also perform essential tasks in the body) and alters their essential functions (Wolf *et al.*, 2017); these include cytochromes, myoglobin and guanylyl cyclase (Kao and Nanagas, 2006; Kondo *et al.*, 2007). During poisoning, many problems can be caused by the binding of CO to these molecules; for instance, as cytochromes are responsible for oxidation and reduction reactions as necessary in the generation of adenosine triphosphate (ATP), their disruption many lead to the formation of oxygen free radicals which will have an adverse effect on normal cellular respiration (Goldbaum, Ramirez and Absalon, 1975). Again, this will contribute to the fatigue and lethargy and headache, etcetera, that an individual feels; binding to other proteins also contributes to muscle pain and explains issues such as incontinence.

There are more explanations as to why the organs of the body, particularly the heart and brain, are vulnerable to the complexities of carbon monoxide poisoning (Prockop and Chichkova, 2007). Predominantly, though, both organs have a high metabolic rate and a significant need for oxygen (Chavouzis and Pneumatikos, 2014). CO also has a high affinity for binding with cardiac myoglobin, which consequently leads to myocardial depression, hypotension and arrhythmia, and ischaemic cardiac changes (Shen *et al.*, 2015). It also disrupts cerebrovascular compensation mechanisms (Kondo *et al.*, 2007).

Binding of myoglobin, which is the primary oxygen-carrying pigment of muscular tissue, is said to occur at an even higher rate than that of haemoglobin (Harper and Croft-Baker, 2004) and can therefore lead to reduced availability of oxygen in the musculoskeletal and cardiac systems, causing pain and weakness. Subsequent arrhythmias, myocardial, mitral valve dysfunction (Gandini *et al.*, 2001) and even infarction across different age-groups, including children, can therefore result. It also contributes to direct skeletal muscle toxicity and rhabdomyolysis.

Guanylyl cyclase is stimulated in the presence of CO. This results in cerebral vasodilation, which is linked with loss of consciousness (Kao and Nanagas, 2006). So, not only are the tissues suffering due to a lack of oxygen but all of the cells in the body suffer due to the metabolic consequence of the hypoxia as a consequence of the binding of CO to other proteins

In a study examining the hypothesis that CO has the potential to inhibit mitochondrial electron transport, which forms part of cellular respiration, in the brains of rats, it was found that prolonged intracellular hypoxia and the resultant acidosis could be a consequence of relatively short-lived episodes of severe CO hypoxia (Brown and Piantadosi, 1992). The authors found that post CO exposure, the mitochondrial oxidation state (affected by the oxidation levels of the cytochrome cells) and metabolite responses indicate that aerobic energy production in the cortex was still impaired after near-total elimination of COHb from the blood, despite the administration of supplemental oxygen; again, this may help explain the prolonged clinical effects of a poisoning event even after the decline of COHb levels (Kao and Nanagas, 2006).

The knowledge about CO instigating cellular hypoxia originated in the nineteenth century. After Bernard suggested that COHb was formed in the bloodstreams of those who had inhaled CO, in 1865 (Reboul *et al.*, 2012). Haldane (Haldane, 1895) reasoned that hypoxia would be the inevitable result of the formation of COHb. His proposal that

hypoxic insult was the central issue has become the accepted explanation for carbon monoxide toxicity (Gorman *et al.*, 2003; Roderique *et al.*, 2015). Haldane's son, however, later went on to experiment with CO and germinating cress seeds and moths. These organisms obviously do not possess oxyhaemoglobin and therefore cannot develop the COHb that would starve their tissues of oxygen. The younger Haldane established that moths are hindered in their movements and the rate of germination in the cress seeds was inhibited by the presence of CO (Haldane, 1927; Roderique *et al.*, 2015). He could, of course, only speculate as to how and why those organisms were adversely affected by CO in that situation, but he did conclude that CO has a 'mortal toxicity' that is *distinct* from its hypoxaemic effects. s

2.1.4. A certain level of exposure/poisoning will correspond to certain symptoms

The haemoglobin-centric theory of CO toxicity accounts for the current therapies of high concentration and/or high pressure oxygen; however, COHb levels do not correlate well with symptoms of poisoning or with clinical outcomes for survivors (Raub *et al.*, 2000; Hampson and Hauff, 2008). (Roderique *et al.*, 2015) postulates that the relationship between COHb levels and symptom presentation should be 'nearly linear', if the mechanism of poisoning is a reduction in available oxygen leading to hypoxia. The theory also fails to explain the cognitive dysfunction that can develop as much as 270 days after injury, when CO may well have long dissipated and symptoms disappeared (Hu *et al.*, 2011). It is understood that diagnosis of CO poisoning is primarily based on laboratory findings of elevated carboxyhaemoglobin for confirmation (Chavouzis and Pneumatikos, 2014). This can be despite the well-recognised lack of correlation between COHb levels and severity of symptoms; such results may also take precedence over the history-taking of an individual's symptoms. This could potentially lead to misinterpretation and misdiagnosis arising from the belief that any presenting symptoms could be caused by something other than CO.

Authors do agree that some categories of people are more vulnerable to the effects of carbon monoxide poisoning. Infants, pregnant women, older adults and those who suffer from existing respiratory and cardiac conditions are more likely to suffer more serious complications at lower exposure doses of carbon monoxide, due to increased circulatory and respiratory effort and comparatively high metabolic rates (Townsend and Maynard, 2002; Harper and Croft-Baker, 2004). Those with coronary heart disease or chronic obstructive pulmonary disease are more susceptible to CO's effects (Chavouzis and Pneumatikos, 2014). Older adults are more prone to already have another condition which would account for such symptoms (and so carbon monoxide poisoning may not be suspected); they may also have less physiological reserve, so a poisoning event may cause more problems

(Harper and Croft-Baker, 2004). They may also be liable to stay indoors, more be housebound and during cold weather more, so that they tend to spend a considerable amount of time in a poorly ventilated room in their home.

The HPA (Health Protection Agency, 2011) add that these symptoms are more common in those over 40 years of age, with the older adult population, children, and pregnant women and the foetus also being at increased risk of harm late NP sequalae have been shown to occur in up to 50% of the patients with >10% COHb and "this happens grossly in unpredicted manner, and although some risk factors have been identified, there is no secure predictive criterion, including COHb levels and seriousness of acute event" (C&P).

Information from the British Heart Foundation (British Heart Foundation, 2016) shows that an excess of seven million people in the UK live with cardiovascular disease (CVD). Of that number, 2.3 million have coronary heart disease. The WHO's figures for safe levels of exposure for those affected by CVD are 30ppm; an increase beyond that number exacerbates symptoms in angina sufferers (World Health Organization, 2010). CO is an acknowledged gaseous air pollutant (World Health Organization, 2008); while it is difficult to single out the differential effects ascribed to ambient CO in the large variance of existing air pollutants, the rate of fatal and non-fatal cardiovascular events is related to air pollution (Shah *et al.*, 2013; Tirosh and Schnell, 2016). We also know that those with respiratory conditions are more likely to suffer exacerbation of their conditions if they are exposed to CO than do those without. Again, these conditions are very common, with one in five people in the UK living with asthma,

COPD, or other lung condition according to the British Lung Foundation (British Lung Foundation, 2016). Much work remains to be done sto establish the extent of the extra burden that inhaled CO could cause these individuals, and how much could be done to minimise the effects of CO on their existing conditions.

2.2. *Current Research in Carbon Monoxides*

There has been comparatively little research conducted in to long-term exposure to Carbon Monoxide in households. The main reason for the prosposed research is to improve the public awareness to long-term exposure to CO. Similar research was conducted by Leigh-Smith (Leigh-smith and Frcsed, 2004) to investigate problem of Carbon Monoxide poisoning within small tents. According to the authors, computer modelling has predicted that a dangerous level of CO could be reached inside tents within 30 minutes.

According to Fisher *et all* (Fisher *et al.*, 2013) there were reports of 348 incidents (880 victims: 334 male, 352 female, 194 sex not stated). Reports of incidents increased from 1986 (1) to 2011 (28). There were 298 deaths (169 male, 124 female, 5 sex not reported). The likelihood of a fatal outcome increased with age for both males and females (28%, 1 – 9 years; 71%, 80 years). The source of carbon monoxide was often a central heating or water boiler (48% of 244 incidents). Many incidents (49%) occurred in private dwellings. However, incidents in caravans, tents, sheds and outhouses had a much higher death rate. If a victim was discovered alive chances of survival were relatively good (87%), even if found unconscious. The estimated duration of carbon monoxide exposure ranged from minutes to years in both fatal and non-fatal incidents. Pets were recorded in 31 incidents (17 died). In 5 cases, carbon monoxide poisoning was identified through illness or death of a pet. Prosecutions were recorded in 49 incidents and at least 7 custodial (prison) sentences resulted, with 34 further convictions resulting in a fine. Charges were preferred against either an installer/maintenance engineer (42%), or the landlord (31%).

2.3 Summary

CO is produced by incomplete combustion of carbonate fuels, such as gas, coal, wood and petrol (De Juniac *et al.*, 2012). In modern domestic settings, energy efficient housing results in more airtightness and fewer draughts, therefore gas appliances that are poorly fitted and/or repaired and maintained can be a source of CO, as can blockages of vents, flues and chimneys (Kokkarinen *et al.*, 2014). De Juniac *et al* (De Juniac *et al.*, 2012)work reported data from COGSS (UK) which showed that fatalities (unintentional, non-fire) occurred from problems with heaters, boilers and cookers, with mains gas accounting for 38.3% of deaths during the period 1996-2007. Unintentional poisoning occurs, unsurprisingly, more frequently in the winter months (Ezratty, Ormandy and Koscielny, 2011; Chun *et al.*, 2012; De Juniac *et al.*, 2012)

Homes with gas ovens where there is proper maintenance have levels that vary between 5-15ppm, compared with 0.5-5ppm where there is no gas oven. Homes with improperly maintained gas ovens may have levels of 30ppm or higher (United States Environmental Protection Agency, 2011). Homes with dangerous appliances. Energy efficient housing results in more airtightness and fewer draughts, but more risk of CO build up

CO is also produced in the body naturally, as a result of the breakdown of haem (Wright, 2002). It is an essential molecule involved in normal cell functions and signalling; it is not merely a poison, but an important 'gasotransmitter' (Roderique *et al.*, 2015). Non-smokers normally have a value of COHb of <2%, while smokers' values can range between 3-12% (Chavouzis and Pneumatikos, 2014). This higher, normal reading can cause difficulty in cases where chronic CO poisoning is suspected; however, there does not seem that smokers experience

the unpleasant symptoms associated with CO poisoning on a normal day-to-day basis, unless they are actually exposed to additional CO.

3. Initial study to investigate CO dispersion between adjoined properties

Public awareness of the risk from CO has increased over the years increased due to various campaigns and work streams, which has led to an increased ownership of CO alarms in domestic properties and resulted in a subsequent increase in requests for a FRS response.

Merseyside Fire & Rescue Services like many other FRS's receiving a call to an incident of this nature will also confirm this guidance and attend the property as a no blue light response. MFRS records show this operational response strategy for 95% of attended incidents resulted in 0ppm reading due to the ventilation of the property releasing any contained CO. Any valuable evidence regarding levels of exposure of potential sources are therefore lost.

MFRS are interested in a potential change in the guidance in relation to the evacuation procedure to enable better understanding and detection of potential sources of Carbon Monoxide inside of the affected properties. To investigate CO build-up in domestic properties this project has made use of three research houses at its Byrom Street Campus that are part of BRE Network, and are used for a variety of building related research topics. These houses were built according to standards from three different time periods namely, 1920, 1970 and 2010. This provide an ideal opportunity to investigate the distribution of Carbon Monoxide across terraced dwellings.

Figure 3.1. Firefighters ventilating the property to make the houses safe.

50 Carbon Monoxide LASCAR data loggers were distributed across the 3 houses. Each logger was set to record CO every 10 seconds during the experiment. The data logger layout is presented in Figure 3.3(Ground Floor) and Figure 3.4 (First Floor).
All interior doors were closed during the experiment and opened by firefighters when ventilating the properties at the end of the experiment. To produce the source of CO in the property 2 portable BBQs were installed in the kitchen on the hob in the 1920 House.
A ToxiRAE 3 CO detector was also placed on the living room and kitchen windowsills, of the 1920 House with the display facing the window to enable real-time CO levels to be observed externally during the experiment.

Fire crews wearing BA and equipped with a ToxiRAE 3 CO detector and communications first entered the 1920's property and took readings within each room, they then secured the property and repeated the process in the other two. Once completed the sources were removed and the property was ventilated in line with general guidance for 10 minutes (assumed non-blue light attendance time).

The findings (Figure 3.2) demonstrate that a source of CO that generated a maximum measured concentration of 320ppm, CO was distributed around the property into the adjacent properties.

	1920		1970	2010
Room	Before Ventillation	After Ventillation		
Kitchen	310	5	11	4
Hall	135	5	9	5
Lounge	164	4	9	4
Landing	182	5	10	4
Front Bed	70	5	17	4
Rear Bed 1	80	5	10	4
Rear Bed 2	80	5	20	4
Bathroom	69	5	9	5

Figure 3.2. ToxiRAE 3 findings.

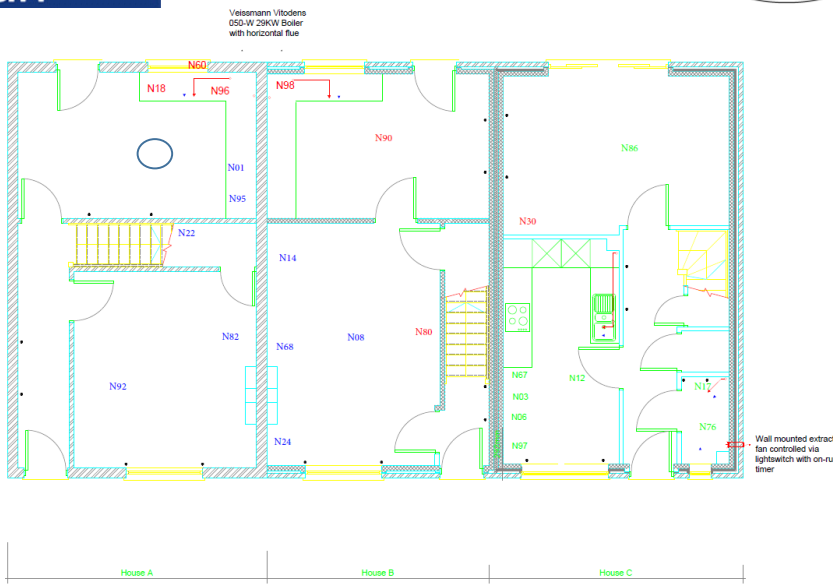

Figure 3.3. Positions of data loggers on the ground floor.

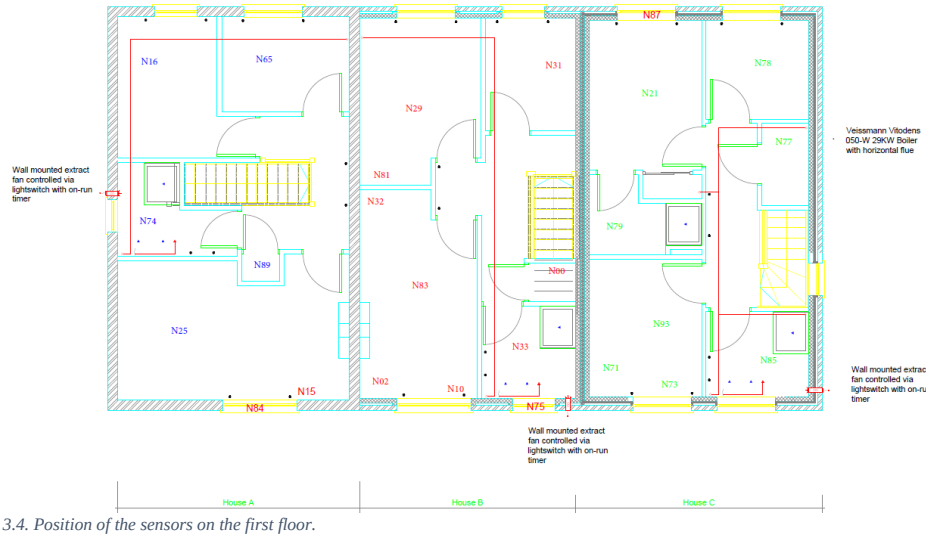

Figure 3.4. Position of the sensors on the first floor.

The Timeline and key events of the experiment are presented below:

11:00- Sensors *in situ*, Doors and windows closed

12:15- 2 disposable BBQs lit in kitchen in 1920 house. All houses evacuated.

12:30- Toxirae in front room reading 6ppm

12:32- Toxirae in kitchen reading 200ppm

13:15- MFRS opened and closed front door to demonstrate someone coming out of their home

13:19- 2 firefighters go into 1920 house to take readings

13:25- 2 firefighters go into 1970 house to take readings

13:20- 2 firefighters go into 2010 house to take readings

13:31- 2 firefighters return to 1920 house to repeat readings (after 10 minutes ventilation)

Figure 3.5. Research Houses with MFRS.

Figure 3.6. Fire Fighters Entering 1920 House.

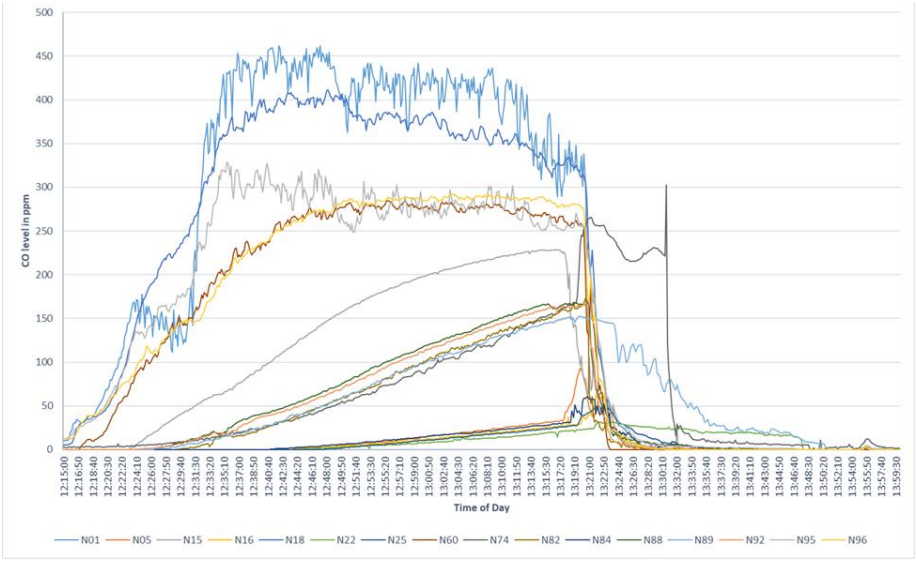

Figure 3.7. CO- Lascar Data Logger outputs for 1920 exemplar house.

Figure 3.7 is a plot of all the recordings from the data loggers placed in the 1920 house. Data Loggers N01 (462 ppm max) and N18 (411.5 ppm max) presented the highest CO level as there were placed close to the CO source. Data Loggers N60, N95 and N96 were also in the kitchen but in different locations, and showed peak values of 285, 328.5 and 292.5 respectively. The variation is caused by the diffusion of the CO. Looking at the time to elevate the Carbon Monoxide, N01 exceeds 50 ppm in 4.5minutes whereas N60 takes 6 minutes and 50 seconds. The stair case reached a maximum of 31.5 ppm, which demonstrates that if an CO alarm were located in the wrong place it would not necessarily identify the life threatening level of CO. In addition, highlights that even if appliance generating the CO is switched off a different part of the house could still have potentially a life threatening level of CO.

Commented [KP2]: What is a house is not ventilated following a CO alarm event to allow FRS to determine the cause but someone is unwillingly left in the house?

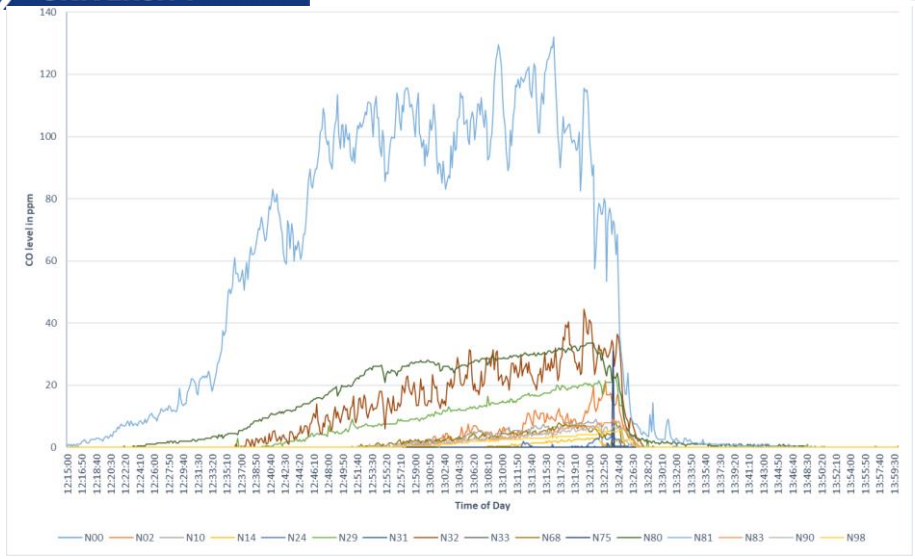

Figure 3.8. CO- Lascar data logger outputs for 1970 research house.

Figure 3.8 shows the data loggers reading from the adjoining 1970 house. The highest CO level was detected by N81 (132 ppm which took 1hour 1minute 30 seconds) and reached the alarm level after 20 minutes and 30 seconds. However, none of the other data loggers placed in the 1970 house exceeded the alarm threshold although they were trending all upwards.

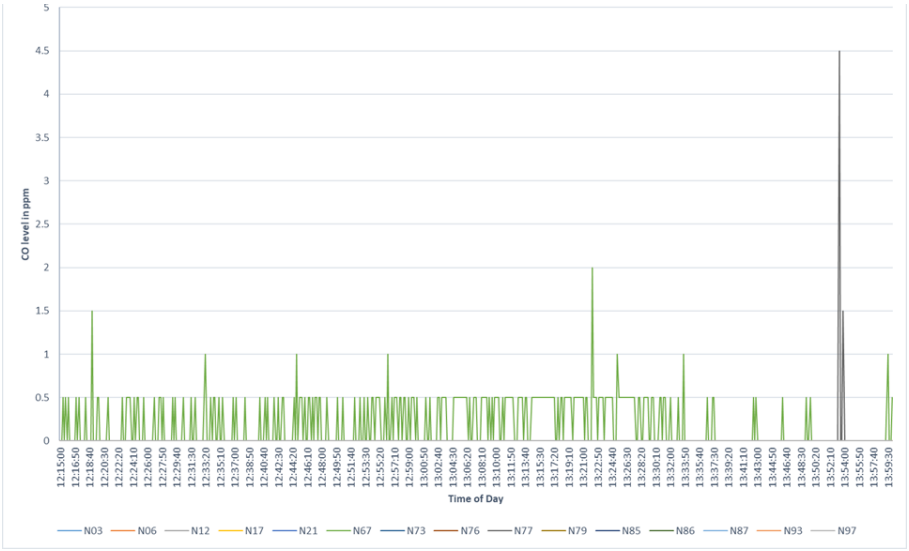

Figure 3.9. CO- Lascar data logger outputs for 2010 research house.

Figure 3.9 shows the data logger results from the 2010 research house. The level of CO did not exceed the dangerous level of exposure.

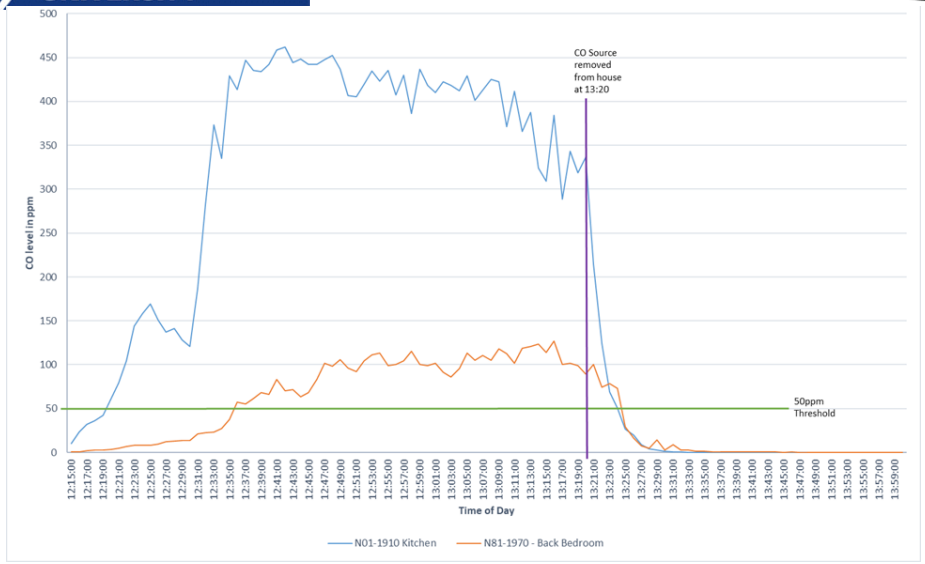

Figure 3.10. CO- Lascar data logger outputs for the highest readings in 1920 and 1970 houses

Figure 3.10 demonstrates the time taken to ventilate properties to a safe level of CO after the sources were removed from the property. It took 8 minutes to reach a level below 6ppm in both the 1920 and the 1970 houses. This demonstrates the initial concept that if the occupier switches off the source and ventilate the property while awaiting for the emergency services to arrive the houses would may well not any presence of CO.

4. National study to investigate chronic low level exposure to Carbon Monoxide

The initial study conducted in 2011 between Liverpool John Moores University, West Midlands Fire & Rescue Services and Merseyside Fire & Rescue Services resulted in around 32,000 sets of data being captured, for academic credibility this data was cleansed and reduced to 22,000 data sets. Within all the evidence that was held within the data one point stood out more than any other, out of 22,000 only 49 readings were recorded over 0ppm.

This prompted a new approach as demonstrated by Croxford (Croxford, Leonardi and Kreis, 2008), and led to a second phase, which introduced the use of Lascar Data Loggers (suggested by CogDem), which were programed to take samples at 1 minute intervals for a period of 2 to 3 weeks. Properties selected for the study, selected from previously visited locations during Phase 1, with a maximum reading of 0ppm during the Home Fire Safety Check visit by Fire and Rescue Service personnel. The obtained results were unexpected since the initial visits provided a 0 ppm reading, all of the data loggers identified CO occurrences above 0 ppm.

Following from previous study, this research was initiated to attempt chronic exposure of Carbon Monoxide nationally. Different fire services teams, coordinated by MFRS participated in this study by distributing CO Loggers for data collection to monitor low-level exposure to Carbon Monoxide. Each participant received a CO alarm donated by CogDem for participation in this study as well as to make sure that each property has a working alarm in case of acute exposure to CO. Data Loggers were left in selected properties between two and three weeks. Data was recorded every 5 minutes to enable capturing duration of occurrences for increased level of CO. Figure 4.1 presents the highlighted regions of data loggers recordings across United Kingdom. Participants for this study were selected by individual fire and rescue services in the selected regions.

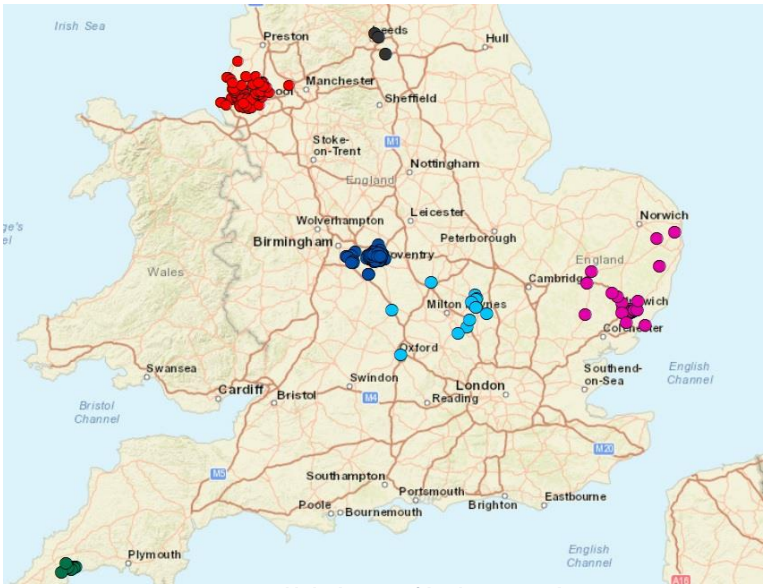

Figure 4.1. Highlighted regions of data loggers recordings

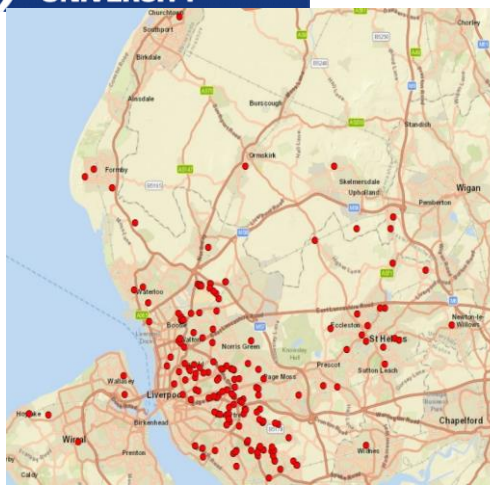

Figure 4.2. Data logger distribution across Merseyside.

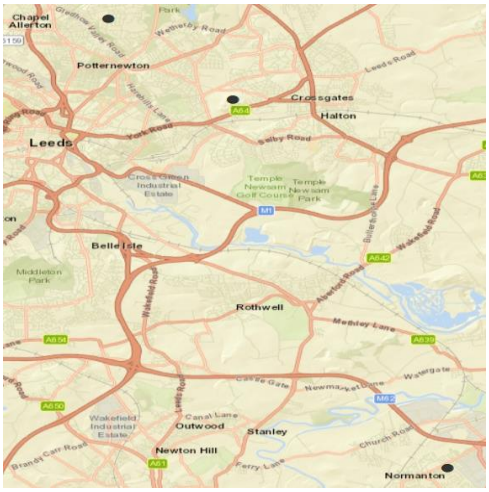

Figure 4.3. Data logger distribution around Leeds.

Figure 4.4. Data logger distribution around Ipswich.

Figure 4.5. Data logger distribution around Bedford.

Figure 4.6. Data logger distribution around Cornwall.

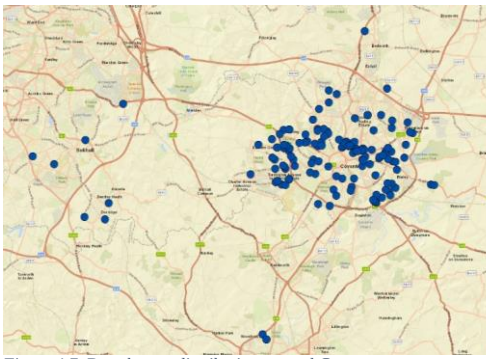

Figure 4.7. Data logger distribution around Coventry.

Figure 4.2 -Figure 4.7 shows the distribution of the data loggers across individual regions. The majority of the study was conducted in Liverpool area and Coventry area owing to the availability of the fire and rescue teams. Data analysis algorithm was written to categorise obtained results in to the CO doses according to WHO guidelines. The created algorithm explore data and analyse different periods namely 24hrs for readings above 6.1ppm and less than 8.6ppm, 8hrs for readings above 8.6ppm and below 30ppm and 1hour for readings above 30ppm. In addition, interventions were counted for each property to investigate if detected occurrences was the single occasion or repeatable event.

Figure 4.8 and Figure 4.9 show example of raw data loggers. In both cases in can be demonstrated that the level of 30ppm was achieved multiple times during the measured period.

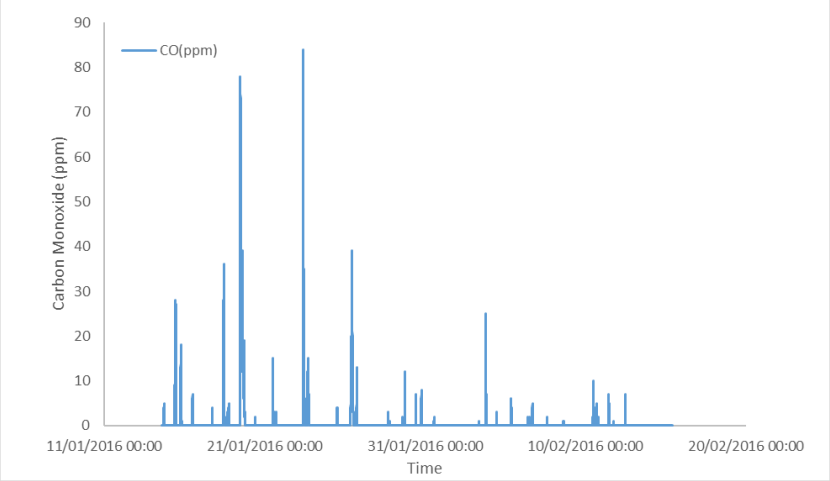

Figure 4.8. Example of raw data from first logger demonstrating readings above 30ppm.

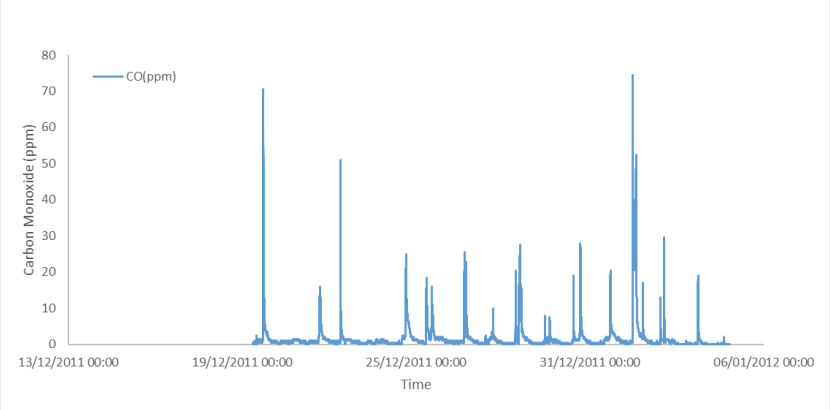

Figure 4.9. Example of raw data from second logger demonstrating readings above 30 ppm.

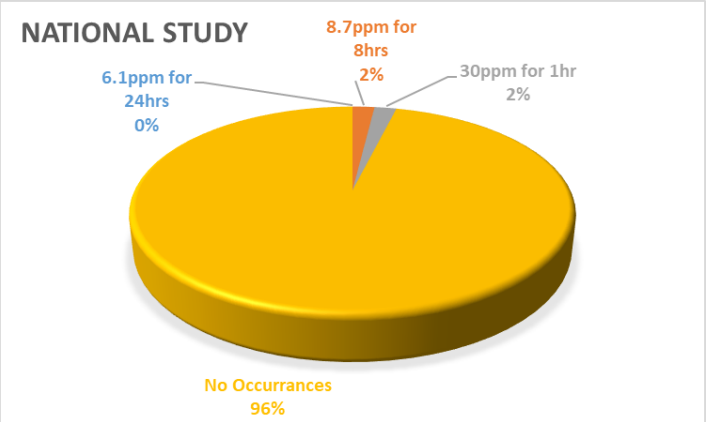

Figure 4.10. National Study main findings from 519 data logger readings.

According to Office for National Statistics (Office for National Statistics, 2017) there were 27.2million households in the United Kingdom if which it is estimated that 7.6million are 1 person households, 9.5 million – 2 people households, 4.4 million – 3 person households, 5.7million- 4 people or more households. Looking at the logger data results in Figure 4.10 it has been found that 4% of 521 households were identified with a significant exposure to CO level according to WHO guidance. This suggests that there could be 1 million households in the UK with increased levels of Carbon Monoxide.

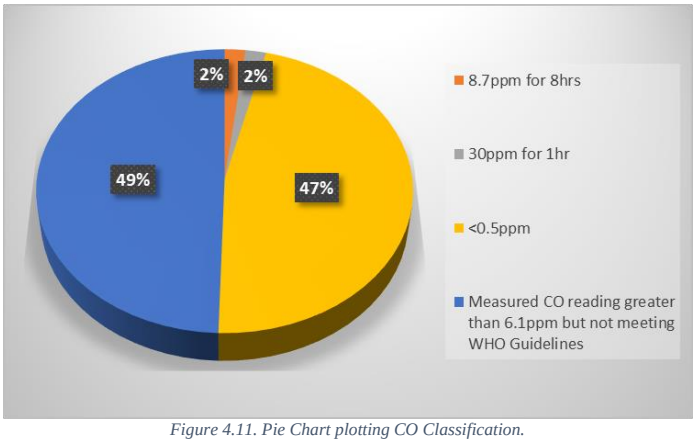

Figure 4.11. Pie Chart plotting CO Classification.

Figure 4.11 demonstrates data loggers CO classification to demonstrate that although 4% of total readings exceeded WHO guidelines. It was found that further 49% of households demonstrated an intermittent level of Carbon Monoxide above 6.1ppm but did not comply with proposed exposure duration suggested by WHO. This could potentially be a concern, for numbers of the public who are house bound. Table 4 highlights numbers of data loggers distributed across selected areas.

Table 4. Distribution of the data loggers across the United Kingdom.

Location	Loggers Distributed
Merseyside	265
Leeds	26
Ipswich	30
Bedford	29
Cornwall	10
Coventry	161

Figure 4.12 - Figure 4.14 show results of low level Carbon Monoxide exposure that meet WHO guidance in the areas where those occurrences were detected.

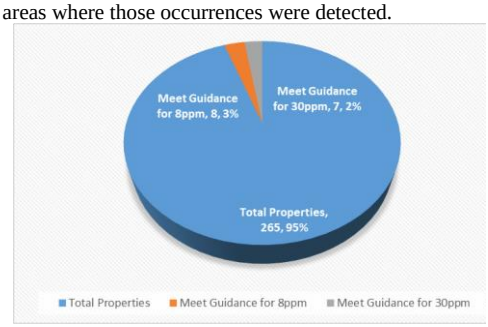

Figure 4.12. Liverpool main findings

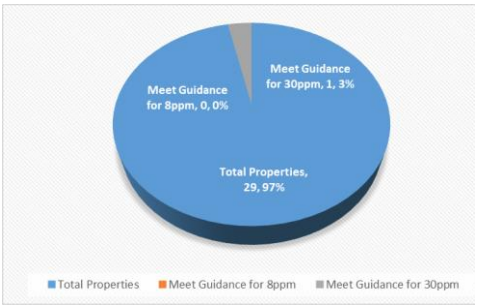

Figure 4.13. Bedford Main Findings

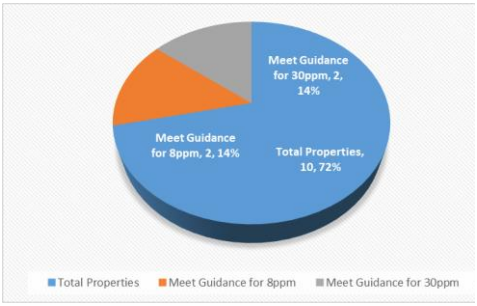

Figure 4.14. Cornwall main findings

5. Conclusion

5.1. Conclusion

The conclusion is that the current response plan is not fit for purpose in regards to responding to a CO incident. The guidance provided eliminates all evidence of CO prior to FRS arrival, so there is no value in the crews attending and it unnecessary increases the organisational road risk. Additionally from a corporate liability perspective it involves advising occupants to enter a potential room of origin and exposing themselves to a possible fatal dose of CO.

These findings led to two options available to inform our change in response:

Option 1 - Produce a revised action plan to provide advice to the occupier which is in line with national guidance from the Council of Gas Detection and Environmental Monitoring (CogDem) and additionally advise the occupier to request a Gas Safety Check through the Gas Safe Register, no MFRS response required.

Option 2 - Advise occupants to turn off any cooking appliance if in use, vacate the property immediately, closing the door behind them. Mobilise an appliance whose crew can carry out a meaningful analysis of the property. Readings taken can then be passed to the gas engineer by the crew or occupant to assist with the identification of the source. Crews should also check adjacent properties if occupied and use the opportunity to complete additional HFSC's whilst in attendance.

5.2. Research Impact

The results of this research has led to an early day motion being raised in Parliament by Margaret Ritchie MP (SDLP, South Down) sighting our work as the main driver for the widening and amendment of that regulation to include all rented property, private and social regardless of fuel source, this motion has now got ministerial approval from Barry Sheerman MP, and the housing legislation for Smoke and Carbon Monoxide alarms within the rented sector (The Smoke and Carbon Monoxide Alarm (England) regulations 2015) is due to change to reflect the recommendations raised.

5.3. Research Limitations

The original concept of project estimated number of logging files as 2,500. Unfortunately, due to austerity measures fire and rescue services did not have a capacity to complete as many loggers as required this was mainly affected by the operational time to recall loggers from properties.

5.4. Further work

This study unveils that exposure to low-level carbon monoxide outside of WHO guidance could potentially affect public in United Kingdom. It would be beneficial to expand the study across additional fire and rescue services. The bigger study can be expanded by involving the other institutions that are currently exploring Carbon Monoxide studies.

Further work could be completed to investigated different logging systems to reduce the operational time required to collect the loggers from properties.

The initial part of the study identify that the type of property could take a major role in the Carbon Monoxide distributing time across the property. This could be further investigated to provide a guidance on ventilation requirements for properties based on the type, year of build and construction.

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