Neutral Citation Number: [2015] EWHC 2264

Case No: 2MA90124

IN THE HIGH COURT OF JUSTICE
QUEEN'S BENCH DIVISION
MANCHESTER DISTRICT REGISTRY
"SONAE GROUP LITIGATION"

Liverpool Civil and Family Court
35 Vernon Street
Liverpool L2 2BX

Date: 30/07/2015

Before:

MR JUSTICE JAY

- -

Between:

MRS SUSAN SAUNDERSON & OTHERS	**Claimants**
- and -	
SONAE INDUSTRIA (UK) LTD	**Defendant**

- -
- -

Mr Michael Redfern QC, Mr Pepin Aslett and Miss Alice Dobbie (instructed by **Camps Solicitors**) for the **Claimants**
Mr Michael Kent QC and Mr Michael Jones (instructed by **Clyde & Co Claims LLP**) for the **Defendant**

Hearing dates: 3rd – 5th, 8th – 12th, 15th – 19th, 22nd – 26th June 2015

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Approved Judgment

Mr Justice Jay:

INTRODUCTION

1. This group litigation involves numerous similar claims for damages for personal injuries, in the torts of negligence and public nuisance, brought by 16,626 Claimants arising out of a major fire at the Defendant's particle board manufacturing plant at Knowsley Industrial Park, Kirkby commencing on Thursday 9th June 2011, at approximately 17:30. The seat of the fire was bunker no. 1 in the Woodyard building at the plant, but the fire spread to all six bunkers in the building, causing a very substantial plume of smoke, fumes, associated chemicals, and particulate matter to issue forth into the surrounding area. In due course, most of the flammable contents of the building were consumed in the fire. The Claimants, all of whom either lived or worked in the neighbouring area or near the plant, say that they were exposed to quantities of smoke sufficient to cause them personal injuries: in particular, a range of symptoms variously involving the respiratory tract, the eye, and the skin; and in some cases headache and more general debility. Fortunately, no one has alleged symptoms of any permanence, and it is accepted that these are low-value claims.

2. Since a Group Litigation Order was made in this case by Hamblen J on 12th July 2012, the issues between the parties have narrowed considerably. By its Group Defence filed on 25th January 2013, the Defendant admitted breach of duty in respect of those who might foreseeably suffer injury in consequence of exposure to the smoke. This admission has removed a layer of evidential exploration, and concomitant potential complexity, from the scope of the litigation. Both the existence and causation of actionable injury remain hotly disputed.

3. By Order dated 17th June 2014 the court directed that there be a trial of common issues and of 40 test cases (at a pre-trial review which took place on 5th May 2015 I reduced the number of test cases to 20). The issues scheduled to that Order (being the varied GLO issues for the purpose of CPR Part 19.13) are:

> "1. When did the fire start and how did it spread?
>
> 2. What part of the site in addition to the Woodyard Building was affected by the fire?
>
> 3. What was the quantity of recycled wood in the Woodyard Building and any other part of the site affected by the fire when the fire started?
>
> 4. What was the composition of such recycled wood when the fire started?
>
> 5. What other materials were in the Woodyard Building and any other part of the site affected by the fire when the fire started?
>
> 6. For how long did the fire burn/smoulder?
>
> 7. When did the recycled wood/other materials burn in the fire?

8. What was the heat output of the fire over time?

9. What were the meteorological conditions during the fire?

10. What was the geographical spread of the smoke plume during the various stages of the fire?

11. What was the chemical composition and concentration of the smoke, in the geographical area in which exposure is alleged, during the various stages of the fire?

12. What air quality or equivalent standards apply to exposure to the smoke?

13. What, if any, air quality or equivalent standards apply to short term exposure to the smoke?

14. What is the relevance of such air quality or equivalent standards that exist to short term exposure?

15. Does *Rylands v Fletcher* apply?

16. What are the findings from the 40 test cases and how should those findings apply to the issues in the individual cases, including:

(i) the extent of the Claimants' smoke exposure.

(ii) the nature of any injury suffered by the Claimants.

(iii) the diagnostic criteria for any such injury.

(iv) the duration of symptoms attributable to any injury suffered by the Claimants.

(v) the cause of any such injury.

(vi) whether such injury was foreseeable.

(vii) whether such injury was actionable in law.

(viii) the relevance of pre-existing medical conditions.

(ix) the relevance of other environmental factors such as cigarette smoke.

(x) Damages (if any)"

4. Since this Order was made, it is apparent that (a) a number of issues have fallen away, either through redundancy (e.g. issue 15) or agreement (e.g. issue 9), and (b) some of the issues require refinement in the light of the parties' greater understanding of the case and/or the Joint Statements of the experts. It is unnecessary to take time in the reformulation of the common issues in this introductory section of my judgment. The

Skeleton Arguments filed at the beginning of this trial have served to stake out the battleground, and indeed during the course of the 18 day hearing a number of issues either disappeared altogether or the parties' respective positions in relation to them converged. I should also record that at a late stage in this litigation an application was made to vary the GLO issues to enable the Claimants to pursue nuisance and annoyance claims which differed from, or fell short of being, claims for damages for personal injuries, but I refused that application because it came far too late and the Defendant had all along been meeting these claims on the basis that they were solely claims for personal injury damages – see, for example, the original GLO issues as set out in the Order of Hamblen J.

5. My judgment will set out the essential factual background in order to provide sense and context to readers unfamiliar with this litigation, but the main focus of what follows will be on what remains in dispute. In truth, not all of the voluminous evidence I received, either orally or in writing, has been entirely relevant to the matters remaining in issue. Moreover, the oral evidence has been transcribed by Livenote, which may readily be referred to if this case goes further.

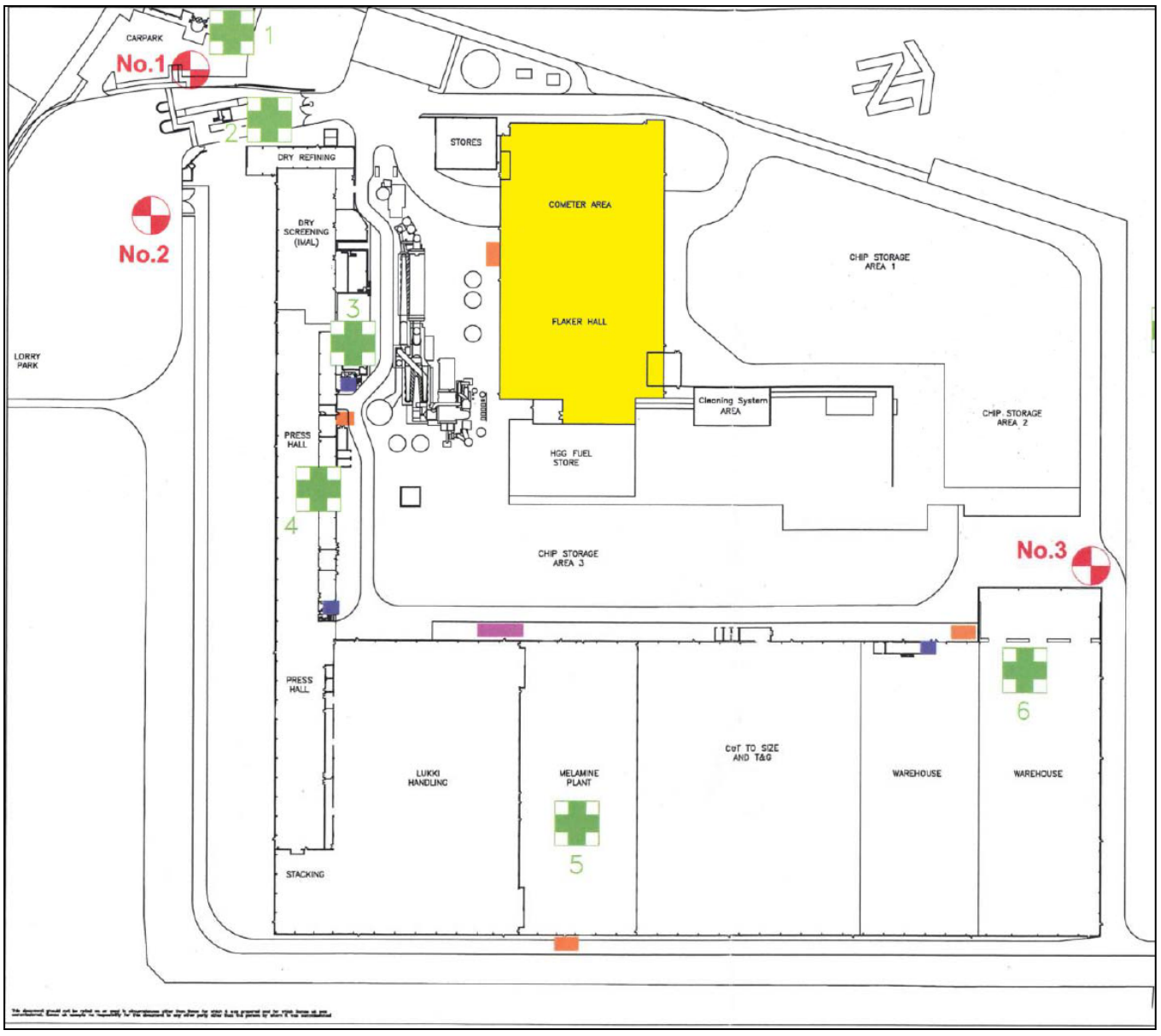
THE DEFENDANT'S PLANT AND *MODUS OPERANDI*

6. The parent company of the Defendant is Sonae Industria SGPS SA, incorporated in Portugal and a global producer of wood-based products. The Kirkby plant opened in July 2000 and ceased production on 14th September 2012. At the time of the fire, the plant operated for 24 hours a day, 7 days a week, and produced approximately 400,000m³ of chipboard product a year. Back in 2000, virgin wood was mainly used, but by 2011 the Defendant's evidence was that 99% of the raw material was recycled wood – with a lesser moisture content and a greater degree of contamination from sundry extraneous matter such as plastic, metal, glass, concrete and general detritus. The use of recycled wood also magnified the inherent fire risk consequent on the storage of a dry, dusty product in a relatively closed environment, and I heard evidence of a significant number of fires before the incident presently under scrutiny. In my view, it is unnecessary to examine the quantum of that risk, and the reasonableness of the Defendant's steps to minimise it, because breach of duty is not in issue.

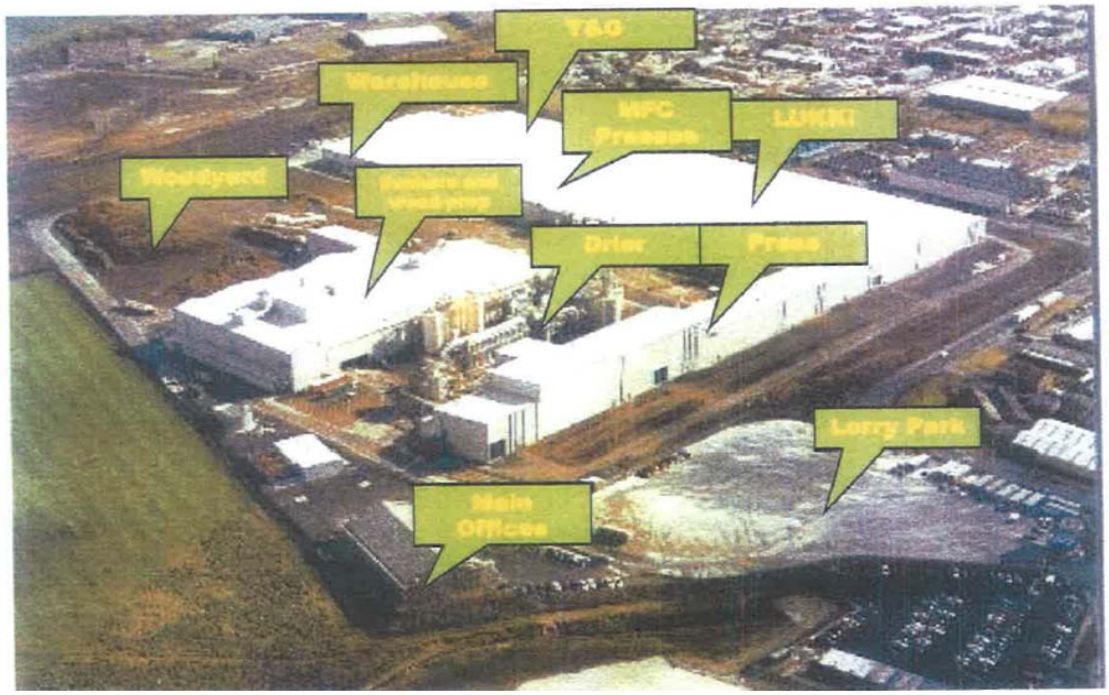
7. The parties called a number of witnesses to explain the workings and operation of the plant. By way of summary, and avoiding matters of controversy at this stage, external suppliers delivered the recycled woodchips to the plant in vehicles variously described as lorries, trucks or wagons. The capacity of these vehicles was up to 25 tonnes, but on average was in the region of 22 tonnes. Although the plant maintained a continuous operation throughout the week, the vast majority of deliveries took place during normal working hours with only around 4% of the total of 386 deliveries a week at weekends. According to the evidence of the Defendant's quality control manager, Mr Mark Callaghan, incoming deliveries were weighed at the weighbridge and then directed to any one of three areas where, after visual inspection and the possibility of rejecting sub-standard consignments, the loads were taken off the vehicles to be introduced in due course into the Defendant's processes. Aside from the external yard where considerable quantities of unprocessed woodchip may be seen on a number of photographs, the loads were either removed from the vehicles using

massive hydraulic, tipping devices called Cometers, or were more straightforwardly unloaded onto the walking floor of the woodyard building. Thereafter, the loads underwent an initial cleaning process (the nature of which varied depending on the pathway taken) before woodchips (by now measuring between 1 and 80mm) were transferred, primarily by overhead conveyors, into any one of six concrete bunkers which were in the same building. These bunkers were contained in three separate silos aligned across the woodyard building in pairs of two. Simultaneously with the deposit of woodchip into the bunkers, this material was being withdrawn via a similar conveyor process (involving the use of a large screw at the bottom of each bunker) to be subjected to further cleansing and manufacturing processes "upstream".

8. The diagram appearing below clearly depicts the general layout of the plant. The construct I am describing as the woodyard building comprises the Cometer area and the Flaker Hall; the external yard is marked on the plan as Chip Storage Areas 1 and 2. Given that the fire started in one of the concrete bunkers in the woodyard building, it is unnecessary to describe later stages in the Defendant's processes culminating in the manufacture of the finished product, namely sheets of chipboard.

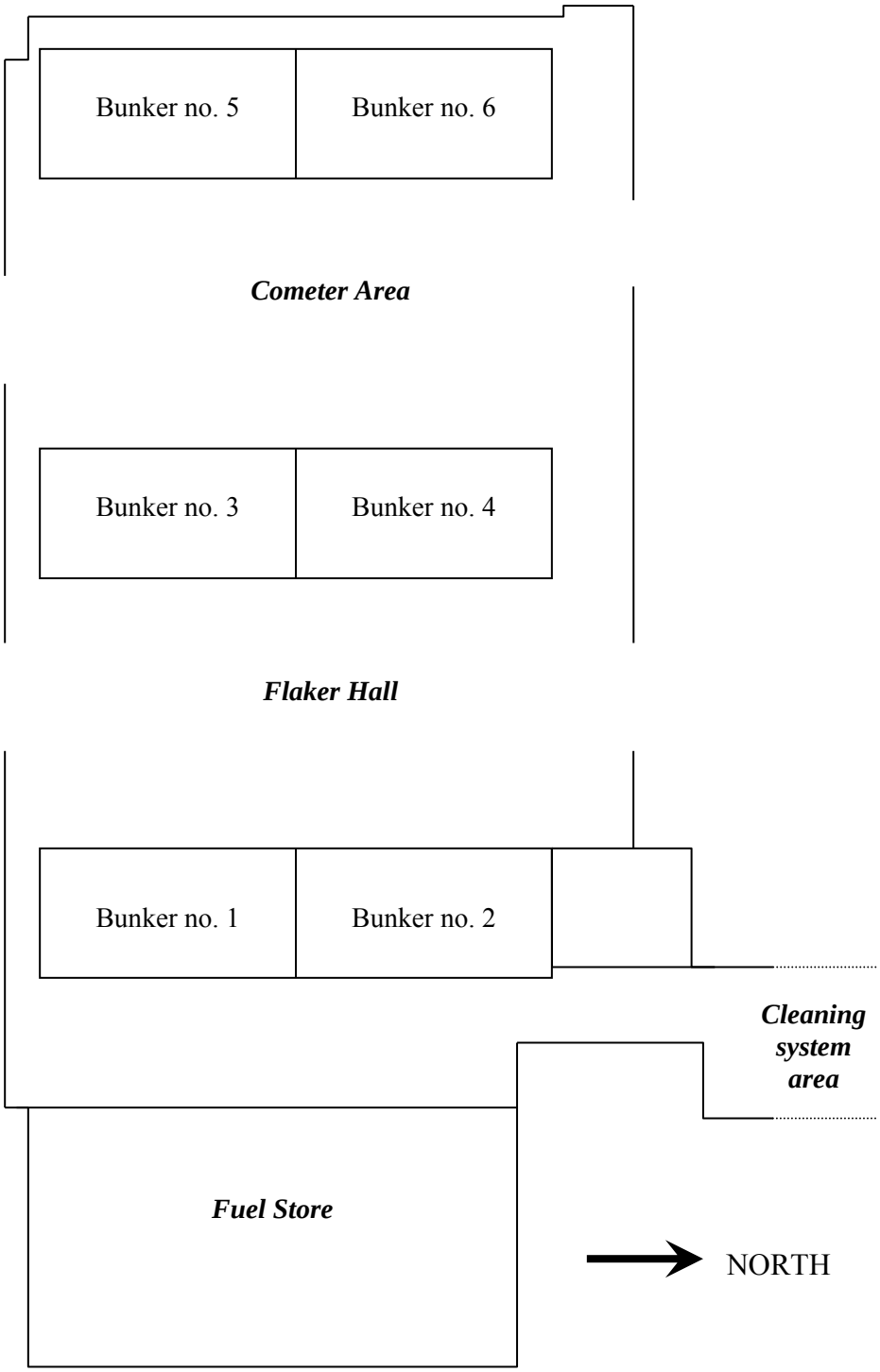


A labelled photograph appearing below shows the external configuration of the plant more naturalistically, although the position and layout of the woodyard building is less clearly depicted. This photograph will be better seen on an electronic version of this judgment.



9. I heard a considerable amount of evidence about the Defendant's quality controls (the efficacy of which was hotly disputed by the Claimants' witnesses), cleaning procedures and quantities of dust. Given the evidence of Mr Gregory Butler, which I consider in more detail below, it is unnecessary for me to address these peripheral disputes. I certainly have the impression of a plant which, at times, was dry and dusty, and the management of which may well have failed to take proactive steps to analyse the fire risk and to address it. However, these issues do not warrant close analysis, nor does the quite general and unspecific evidence about "dressed" loads and degree of contamination. Ultimately, there is convincing quantitative evidence upon which I am able safely to rely.

10. Returning to the layout of the woodyard building, the diagram appearing below, drawn by Dr Jowett, provides a basic schematic pictorial of the position of the silos and bunkers in relation to the Cometer area and the Flaker hall:

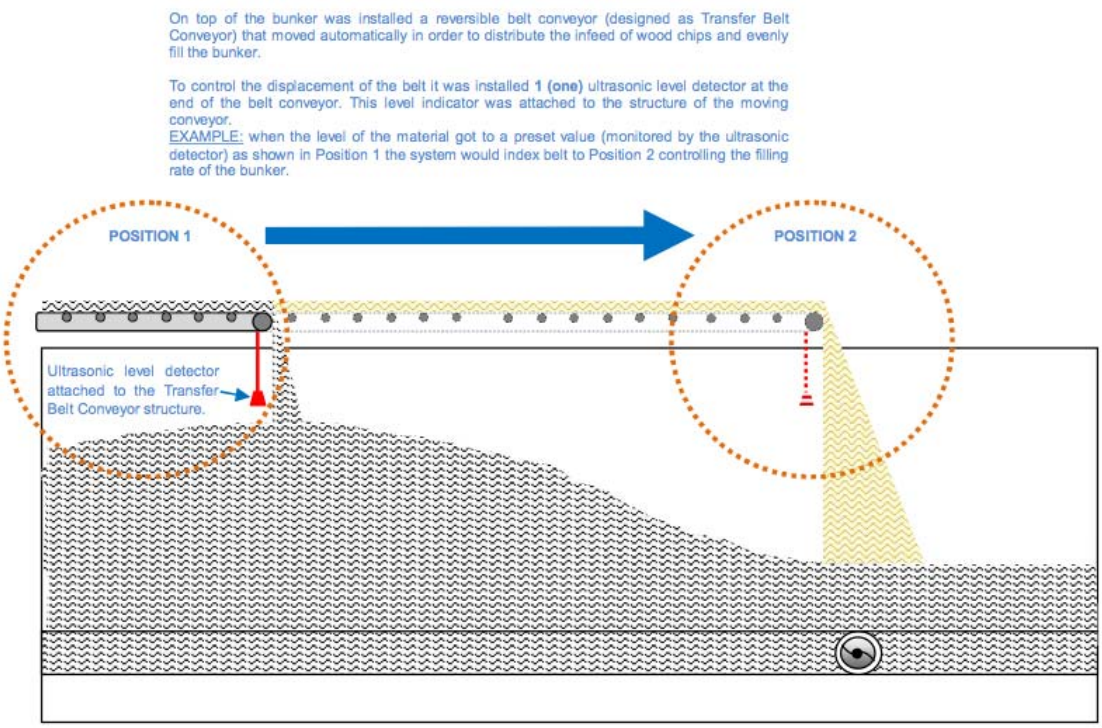


Each bunker was 25m in length, 12m wide and 12m high. Each silo was 50m long and was split into two by means of a 400mm thick dividing wall, made of reinforced concrete. Along the base of each bunker were "letterbox" or slot openings each 970mm deep.

11. The overhead conveyor system was largely automated, and was controlled by a SCADA system running on computer screens in various locations in the plant. There

was also a spark detection or "fire-fly" system intended to provide early warning of combustion within the bunkers, and elsewhere.

12. Appearing below is a diagram, furnished by the Defendant in late April 2015, illustrating the deposit of wood into the bunkers:



The legend explains the position but further explanation is required. The ultrasound level detector (the existence of which was revealed to the Claimants only very late in this litigation) was designed to measure the distance between the conveyor and the level of woodchip directly underneath. When the sensor detected that the level had attained a pre-set value, the conveyor moved along the bunker to another position before further deposits were effected. In this way the bunkers were filled, but not in such a manner that the woodchips were ever horizontally aligned within the plane formed by the tops of bunkers. The woodchips formed peaks and troughs. Had an experiment been carried out to ascertain the maximum capacity of the bunkers through this filling system, one would culminate with a pile of woodchips describing a "Toblerone" shape, with the peak at a level which matched the pre-set value on the sensor. Subject to some doubt as to the angle of incline of this tent shape, the maximum tonnage of each bunker calculated on this basis would have been in the region of 3,600 Te (the Claimant proposes 3,696 Te). However, this theoretical perfection could never be attained in practice, for several reasons. First, operational reasons within the plant would dictate the state of the bunkers, and there was no need for the bunkers to be as full as possible: as the operators all told me, they needed to be *"healthy"*. Secondly, it is apparent from the schematic at paragraph 12 as above that the system was not designed to create a uniform, even peak – hence the troughs to which a number of witnesses referred. Lastly, the process was dynamic in the sense that the bunkers were constantly being emptied via separate conveyors to enable the woodchips to enter the next stage in the process.

13. The SCADA system recorded the data sent back from the bunker sensors (there was one sensor per bunker) on temporary log files, by definition for a limited period. These files are no longer available. Two issues arise: first, whether the data would in any event have thrown valuable light on the content of the bunkers, as opposed to the distance between the sensor and the "drop area" immediately below; and secondly, whether I should draw an inference adverse to the Defendant flowing from the non-retention of these data. I will be returning to these issues under the heading, "The Quantity of Woodchip in the Bunkers".

14. Until a late stage in this litigation, the moisture content and degree of contamination of the woodchips in the bunkers were in issue, and various parameter values were suggested by the Claimants. However, the supplementary witness statement of Mr Gregory Butler, the Defendant's quality manager, supplies an analysis, via sampling data, of the average moisture and contamination levels over a five month period between January and May 2011; and his evidence, although tested, was not seriously challenged under cross-examination. Moreover, although the moisture values must be envisaged as reasonably accurate, I accept the Defendant's contention that the recorded contamination levels in fact *overstate* the actual levels within the bunkers, because the samples were taken before the first cleaning process. In short, I proceed on the basis of data which cannot be significantly impugned, namely a moisture level of 20% and a contamination level of 0.8%.

A SYNOPSIS OF THE COURSE OF THE FIRE

15. It has been estimated that the fire started at approximately 17:30 on Thursday 9th June in bunker no. 1. The initial activation of the "fire-fly" detection system occurred at 18:57, but the processes were not shut down until just before 19:35. Employees smelled smoke, which slightly later was seen to rise from an area towards the south-western corner of the bunker, and attempts were made to douse the nascent fire using a hose. The Merseyside Fire and Rescue Service ("MFRS") was telephoned at 20:03, and fire-fighting personnel soon arrived at the plant. Apart from spraying water into the bunker, the strategy adopted was to remove as much woodchip material from the bunker as possible in order to reduce the actual and potential fire load. At approximately 03:40 on Friday 10th June a flame was seen to rise, or shoot, from the south-western corner of bunker no. 1, fanning in all directions. The flame was dark in appearance, and was lacking oxygen. Within a rapid space of time flames were shooting out of the top of the bunker, and a MFRS evacuation whistle was blown.

16. Thereafter, the development of the fire was sudden. The amount of smoke increased and the fire was soon spreading along the conveyors at a high level in the building. The senior fire officer within the woodyard building described the event as a "conflagration". By 04:00, although the timings cannot be altogether precise, it seems from the evidence of the fire-fighting professionals on the ground that the fire had spread throughout the whole of the woodyard building. By 06:05, all six bunkers were recorded as being on fire, although it is possible that the ferocity of the fire had already encompassed these structures. At that stage the fire was a raging inferno and thick, black smoke was spewing from the roof of the woodyard building in substantial quantities.

17. According to Dr Jowett's report dated 20th June 2011, frictional heating within bunker no. 1 was the most likely cause of the fire. The sudden escalation of the fire was caused by the collapse or slippage of bulk material within the bunker, leading to the ignition of accumulated pyrolysis products formed in the preceding hours.

18. It is common ground that the fire went through three distinct stages or phases. The first stage started at about 17:30 on 9th June (the precise time matters not) and entailed a smouldering fire propagating slowly outwards from the origin, creating a "nest" of burnt and smouldering material. During this stage the heat that was generated by the process of combustion was sufficient to pre-heat woodchips in the vicinity, but insufficient to ignite the resulting pyrolysis products and cause flame. The first stage concluded between 03:35 and 03:40 on Friday 10th June. The transition between the first and the second stages occupied a number of minutes, and the second stage commenced at about 04:00. This was the fully developed, substantial fire involving in due course all six bunkers and other combustible materials within the woodyard building. Amongst the characteristics of this second stage were flame and black smoke. The fire transitioned into the third stage when the appearance and quantity of smoke reduced, and the residue materials were gradually consumed in a smouldering process characterised by "reverse propagation". The parties are not in agreement as to the timing of this transition (which on any view must be somewhat imprecise), the Defendant averring that it occurred by about 10:00 on 10th June, the Claimants by 14:30. Thereafter, the fire diminished in intensity as the residue within the bunkers was gradually consumed within the smoulder, with the MFRS permitting it to burn out in a controlled manner and not declaring the incident officially "closed" until 7th July 2011. By that point, there was very little left in the bunkers except for ash and similar residue. Despite the extent and intensity of the fire, only the woodyard building was involved.

THE ISSUES ARISING FROM OR TOUCHING ON THE NATURE AND EXTENT OF THE FIRE

19. At this stage, it is convenient to identify with greater salience and precision than was achieved on 17th June 2014 (when the parties' knowledge and understanding of the case and what really divided them was not as great as it now appears) the issues which seem to me to arise for resolution in relation to the matters I have ventured to summarise thus far. These are:

    (1) the quantity of woodchips in the bunkers.

    (2) the remaining fire load, in terms of its constituents and its quantities.

    (3) the timing of the transition between stages 2 and 3.

    (4) the heat release rates for the three stages.

    (5) the emission factors which should be applied to this fire.

    (6) ash and dust generation.

20. The relevance of the fourth and fifth issues needs to be explained. In order to achieve indicative values for the levels of exposure of the Test Claimants to relevant chemicals and particles at various times, the plume modellers require a range of data and information, including the relevant heat release rates and emission factors. The concept of a heat release rate is self-explanatory – other things being equal, the greater the rate, the higher the smoke plume travels and the more widely it disperses. The concept of an emission factor is less straightforward, and a full explanation appears under paragraph 92 below. The basic point is that these variables are fed into the computer programme used by the plume modellers. They also need to know the wind speeds and directions at all material times (these are agreed), and finally they factor into their model other variables such as exit velocities, temperature, diameter and number of buildings (all of which variables are now also agreed). The parties are agreed that emission factors constitute the most important scientific issue in this litigation because they are so far apart in relation to it.

21. Once the plume modellers have run their computer model on the basis of the correct heat release rates, emission factors and other variables, the Test Claimants' levels of exposure to chemicals and particles of interest can be evaluated. In order to ascertain whether any individual Test Claimant may have suffered a potentially injurious exposure, a scientific approach would mandate that the raw exposure level predicted or indicated by the model be compared against known toxicological data for the chemicals and particles of interest. These data, to the extent that they are scientifically robust and substantiated at relatively low levels of exposure, will serve as a guide in determining whether any individual Test Claimant's modelled exposure was above or below what might be described as a threshold level for potentially deleterious health effects.

22. I should make clear that the application of scientific methods to engineering, toxicological and modelling questions cannot be regarded as exhaustively determinative of the key issue which arises further down the line, namely whether, having regard to all the available evidence, I should be satisfied on the balance of probabilities that any individual Test Claimant suffered personal injury in consequence of exposure to the products of the Sonae fire. Submissions were deployed as to the difference between the scientific and legal standards of proof, and I will need to address these in due course. However, at this stage of the analysis I should indicate that in my view purely scientific questions (e.g. the ascertainment of the appropriate heat release rates from given data by applying established formulae) can only be answered by applying scientific methods, within the context always of a civil trial: whereas science may require proximity to certainty, the law does not. To the extent that the heat release rates depend on the quantity of woodchips in the bunkers, I approach the issue by applying the traditional probabilistic standards familiar in civil litigation. To the extent that there is inherent uncertainty or imprecision in any given scientific method, this may be reflected in the overall assessment of whether any individual Test Claimant's case is proved on the balance of probabilities.

23. I have mentioned expert evidence in a number of disciplines, and at this stage I should explain the position in slightly more detail. The Defendant has filed and served three reports and one letter from an expert in mechanical and fire engineering, Dr Alan Mitcheson. His evidence bears on the issues of the transition between stages 2 and 3,

heat release rates and deposition of dust and ash. At a pre-trial review which took place on 5th May 2015, the Claimants sought my permission to file and serve comparable evidence from Dr Phylaktou (I understand that he would also have assisted me on the issue of emission factors), but I refused permission and relief from sanctions, on the ground that it was far too late to adduce such evidence, and the smooth running of the trial would be seriously prejudiced. My detailed reasons appear in a separate transcript. Accordingly, the Claimants have no expert evidence addressing these issues, although they do rely on their modelling expert, Dr David Carruthers of Cambridge Environmental Research Consultants Ltd, to address emission factors and the issue of radiative heat loss away from the smoke plume (if a valid point, this would reduce the relevant heat release rates accordingly). Issues arise as to Dr Carruthers' qualifications to deal with some of these issues. The Defendant has its own modelling expert, Mrs Angela Spanton of Envirobods, and the parties have experts in meteorology (as I have said, their evidence is agreed) and toxicology. In relation to this last discipline, the Claimants rely on the evidence of Professor Alastair Hay, who is an international authority in his subject, whereas the Defendant has called Mr David Shillito, who is not a toxicologist but a chemical engineer with, amongst other things, an environmental background. Mr Michael Redfern QC for the Claimants did not object to Mr Shillito's contributions in this regard, nor did he object to him assisting the court on the issue of emission factors – a matter on which he is qualified to opine, although it might be said that the Defendant has exceeded its permitted quota of one engineer.

24. I have to say that the state of the expert evidence in these technical disciplines is not wholly satisfactory. In particular, I consider that I would have benefitted from more evidence on the issue of emission factors. However, I am not conducting a public inquiry, and I have to do the best I can on the available evidence, remaining loyal to Mr Redfern's warning, albeit delivered in a different context, that judges are not "super-scientists".

The Quantity of Woodchips in the Bunkers

25. I heard a range of evidence of variable quality and precision relating to this issue. The theoretical maximum capacity of the six bunkers was in the region of 4,200 Te (i.e. full to the brim), and the theoretical maximum achievable in deployment of the sensor system was 3,696 Te (the "Toblerone" shape I have previously explained). The Claimants' case is that I should favour a figure in the region of 3,000 Te, making allowances for what was practically attainable; the Defendant's case is that there were 1,550 Te of woodchips in the six bunkers.

26. The vast majority of the evidence bearing on this issue was of a lay, not an expert, nature. The Defendant's mechanical and fire engineer, Dr Mitcheson, has contributed to the issue to a minor extent.

27. The majority of the Claimants' generic witnesses gave somewhat vague, inconsistent evidence on this issue. According to paragraph 26 of the witness statement of Mr Brian Beardwood, his belief was that for reasons of production the bunkers were nearly full at the time of the fire. In oral evidence, however, he could not say how much was in the bunkers, save that the levels were *"healthy"*, which to him meant at

least half full. Mr Brian Hoyles told me that the bunkers were almost always full during the week, and fuller during the week than at weekends. According to paragraph 33 of the witness statement of Mr Michael McNamara, his belief was that bunker no. 1 was totally full at the time of the fire. He was not certain, but believed that the remaining bunkers must have been at least half full *"because they tried to keep them full all the time"*. In his supplemental witness statement Mr McNamara told me that one could view two months' worth of data on the SCADA system, and there were sensors on the bunkers which *"definitely did measure the quantity of woodchips in the bunkers"*. He agreed in cross-examination that his priority was the wet silos (further down the production line) and not the bunkers, and accepted in relation to the latter that he was not sure whether the sensors just measured the height of the drop immediately below them. In re-examination, Mr McNamara said that as far as he could recall, when he was reviewing the SCADA data for past trends, he was looking at the data relating to the mixed-chip silos (i.e. not the bunkers).

28. Mr John Rimmer, a maintenance technician, said in a statement introduced as hearsay evidence under the Civil Evidence Act 1995 that the bunkers were *"pretty full, 65% at least at the relevant time"*. The weight to be given to this somewhat generalised and unspecific evidence can only be very slight.

29. Impressionistic and inconsistent evidence of this nature from witnesses who I would assess as not being particularly reliable falls a long way short of being sufficient to prove the Claimants' case that there was as much as 3,000 Te in the six bunkers. Further, I have already explained why operational reasons would not require the bunkers to be so full, and that the process was dynamic. In any event, other evidence called by the Claimants solidifies my conclusion that the 3,000 Te figure cannot be right.

30. Mr Terence Poulson, who I regret to say was a very poor witness, told me that in the weeks before the fire the bunkers were 85% full to his knowledge. On the night of the fire, he could see what was in the bunkers, and they were 85%, maybe 90% full. Under cross-examination, Mr Poulson expanded on this evidence and said that he checked the level of the bunkers from the top of the gantry, because he was trying to find out the source of the fire. Yet, at paragraph 19 of his witness statement, Mr Poulson clearly stated that he was not on the overhead gantry on the night of the fire, but was there the day before. This is consistent with an earlier draft witness statement which was put to him in cross-examination. Mr Poulson tried to persuade me that he was *"at the top of the stairs"* rather than on the gantry itself. I did not believe him. Another point which weighs heavily against him is that the evidence he adduced to support his claim for damages for personal injuries was inconsistent, exaggerated and in some respects (e.g. the reference in his questionnaire to GP attendances) clearly untrue. I cannot accept Mr Redfern's submission that issues of credibility bearing on Mr Poulson's symptoms must be hermetically sealed from the credibility of his evidence relating to the bunker levels. Mr Poulson well knew that the greater the contents of the bunkers, the greater his prospects of succeeding on his damages claim.

31. Another very poor witness was Mr Michael Moorcroft, who probably started working at the plant as an industrial cleaner and occasional FLT operator on 22nd May 2011. In his evidence in chief, he said *"every time I visited the bunkers, I would say that they were at least 85% full"*. According to paragraph 7 of his witness statement, *"I can definitely say that they were full"*. However, it emerged in cross-examination that this

witness did not know that there were six bunkers in all. Further, his personal injuries' claim, although only cursorily examined because the whole picture was not available, appeared extremely tenuous on the basis of the probable exposures to which Mr Moorcroft was able to attest, and moreover his diagnosis of conjunctivitis on 10th May 2011 could have had nothing to do with the fire or his work at this plant.

32. I cannot conclude that because these two witnesses gave at best unreliable, at worse untruthful, evidence it must follow that the Defendant's case is right. However, evidence of this quality has the tendency to undermine the Claimants' case that the levels were as high as 3,000 Te, or even 85% of that figure.

33. On 14th June 2011 Dr Paul Jowett of Dr J.H. Burgoyne and Partners LLP, appointed to investigate the causes and circumstances of the fire by the Defendant's insurers, held a preliminary meeting with Rodney Mitchell, its Chief Technical Officer, Nigel Graham, its Managing Director, and other senior employees of the company. Dr Jowett was given contradictory information as to the contents of the bunkers. The Defendant disclosed Dr Jowett's notes of this meeting, and others, extremely late in the day but ultimately I conclude that nothing turns on that. His notes are a Curate's egg from the Claimants' perspective; and, more importantly, they are reliable. Dr Jowett was informed of the identity of employees he might interview in the course of his investigation. One of those employees was Mr Thomas Pybis.

34. The Claimants called Mr Pybis to support their case on this issue. He had been working as a Cometer operator in the woodyard building since approximately June 2008. It is highly relevant that he was not asked to provide a witness statement until this year. On 16th June 2011 he was interviewed by Dr Jowett in relation to what he knew about the possible causes of the fire and the quantities in the bunkers. Dr Jowett kept a contemporaneous manuscript note and in my judgment was a particularly impressive witness: moderate, measured, and astute to answer the questions put to him. The reason why Dr Jowett took the trouble to speak to Mr Pybis was because he was the last person to check the contents of the bunkers at approximately 18:50 on 9th June. Mr Pybis gave Dr Jowett valuable information as to the profile of the woodchips in bunker no. 1, and the manuscript diagram Dr Jowett was able to draw was clearly based solely on that intelligence. When Dr Jowett's detailed notes were put to Mr Pybis in cross-examination, he agreed that they were largely accurate, save as to the timing of the last inspection (in relation to his dinner break) and the tonnages Dr Jowett recorded in relation to the bunkers. The relevant section of Dr Jowett's notes reads as follows:

"Bunker 1	I estimate 200-250 tonnes a.t.o.f. [at time of fire] (i.e. about ½ full)
2	been out of use – prob[lem]s with screw ∴ ~ 100 tonne
(sawdust)	~ 200 T
(purchased chips)	~ 300-350 T
Recycled wood 1 (S)	~ 350 T

Recycled wood 2 (N) ~ 350 T"

35. It should be explained that Mr Pybis was using the names or labels for these bunkers which were a throwback to the days when the plant took in virgin wood, sawdust, purchased chips and some recycled wood. The two recycled wood bunkers are numbered 5 and 6 on the plan (see paragraph 10 above). The aggregate of these tonnages is 1,500 – 1,600 Te, and Dr Jowett has taken the mid-figure of 1,550. Subsequently, Dr Mitcheson has used the self-same metric to base his heat release rate calculations.

36. Mr Pybis' oral evidence was that the bunkers were habitually kept at a *"healthy"* level, which in his view was anything in excess of 50% of capacity. He said, as did other witnesses, that the bunkers would tend to be full on Thursday and Friday to support weekend production. In cross-examination, he agreed that the SCADA system did not indicate whether a bunker was full; it would merely show the operators what was happening directly below the drop level. It was for this reason that visual assessment of the bunkers, which entailed climbing up the stairs onto the gantry, was the best way to evaluate the position, and was therefore habitually undertaken. As regards Dr Jowett's notes, Mr Pybis did not accept that he would have told him the levels in terms of tonnages. He did not in fact know these. Instead, he would have told him what the levels were in terms of fractions or percentages. Mr Pybis agreed that he might have said that bunker no. 1 was half full, but he would not have said that there was less in bunker no. 2. In fact, his recollection was that bunker no. 2 was 75-85% full. He was less sure about that in cross-examination. In his opinion, all the bunkers had *"healthy"* levels, although there were different levels in each of the bins.

37. Mr Redfern urged me to treat Dr Jowett's hearsay evidence, both that contained in his notes and his oral account from the witness box, with considerable caution. The notes were not checked for accuracy by Mr Pybis, and the possibility for misunderstanding arises. In principle, I am content to adopt that cautious approach in reaching appropriate conclusions about this important seam of testimony. However, having seen and heard both Dr Jowett and Mr Pybis, I am confident that the former's notes are accurate.

38. Mr Pybis gave the impression of trying too hard to persuade his listeners that he would not have mentioned tonnages to Dr Jowett. I am sure that he did, for a number of reasons. He was a reasonably able witness who told me that he was passionate about his work. He had been working in the Cometer hall for a number of years, and must have known the capacities of these bunkers in terms of tonnages. In answer to my questions, he agreed that he heard others mention 500 tonnes. When pressed in cross-examination, Mr Pybis let slip that he *rarely* mentioned tonnages; he felt more confident using percentages. It follows that he sometimes mentioned them. Finally, Dr Jowett's unchallenged evidence was that he simply wrote down what he was told. He had no reason to do otherwise, and it is clear from other sections of his notes that he took care to ensure that he had understood Mr Pybis' explanations.

39. My firm conclusion that Mr Pybis told Dr Jowett that there were approximately 1,550 Te of woodchips in all six bunkers does not prove that the former was right. Mr Redfern makes the points that the Defendant is relying on the evidence of a labourer, and that an international company of this stature should be able to adduce more cogent evidence in a case of this importance. I will deal with Mr Redfern's second

argument later, but as for the first I disagree with his somewhat dismissive assessment of his own witness. Mr Pybis had worked his way up from being an industrial cleaner, and his competence and experience are not in question. He was the last person who in fact witnessed the levels in these bunkers. In my judgment, his experienced assessments were reasonably reliable, and would have been at least as robust as anyone else's. I understand but cannot condone Mr Pybis' reasons for wanting to backpedal. He was not credible in that respect, but the reliability of what he told Dr Jowett has not been undermined.

40. Independent evidence as to the quantities in the bunkers comes from the contemporaneous logs completed by fire officers of the MFRS. However, this evidence is hearsay, somewhat contradictory and unreliable. MFRS's sources are unknown.

41. The Defendant's generic evidence of fact throws limited additional light on this issue. Mr Mark Callaghan told me that the bunkers were usually approximately 40-50% full (200-250 Te), which evidence is consistent with what he told Dr Jowett in June 2011. Mr Callaghan said that the Defendant was planning to stop production on Monday 13th June for at least three weeks. This evidence came out the blue, and too late in the case for other witnesses to be questioned about it. As I have said, other employees told me that the bunkers would tend to be fuller towards the end of the week. I understand Mr Redfern's concern about the circumstances in which this evidence has seeped into the case, but I believe that Mr Callaghan was a reliable and impressive witness who did not enter the witness box in order to mislead me. Moreover, in my view other former employees called by the Claimants were giving me, at best, a generalised impression of the Defendant's operation, not what was in fact happening on this particular day.

42. In any event, a shutdown on Monday 13th June might well have precluded there being *over* 3,000 Te in the bunkers, but does not necessarily contradict a total tonnage in the region of 2,500-3,000. I say this because Mr Callaghan told me that approximately 700 Te were withdrawn per day. On the basis of three days' production (10th – 12th June inclusive), and the necessity to leave the bunker screws safely covered, the arithmetic speaks for itself. On the other hand, the impending close-down is a pointer which slightly favours the Defendant and serves to neutralise the Claimants' point that as the weekend approached the bunkers tended to be fuller.

43. Mr Callaghan also assisted me with the workings of the SCADA system. He explained that the purpose of the sensor was to monitor the height of the woodchips so that they did not block the conveyor. The icon on the computer screen would then give an indication in the form of a percentage of how much was in the bunker overall. The temporary log files containing this information were probably kept for around three months, but this witness was not sure. He said that a "PLC engineer" would be needed to go back any further. However, he was clear in his evidence that he never used the computer monitoring system to evaluate levels, because there was no need to do so.

44. Mr Callaghan also told me about a shift handover log which contained information about the bunker levels; they were completed by the shift managers on the basis of information from operatives. This was another evidential revelation: no such logs have been disclosed, or mentioned in the Defendant's disclosure lists. Their purpose

was to brief the oncoming shift, and the same information would also be used at monthly meetings. In answer to my question, Mr Callaghan told me that the shift manager on the night was Mr Steve Sharkey. He would have ended his shift at 22:00, and would have *"gone round the area"* maybe once during the course of the shift. I was given no explanation as to why he was not called to give evidence about the bunker levels, but it is clear that he did not speak to Dr Jowett about them and it is probable that what he knew about bunker levels that night came from Mr Pybis. Furthermore, I draw the reasonable inference that it is highly unlikely that any shift handover log would have been prepared and completed on the night of the fire at or near to 22:00. There were other, more pressing concerns.

45. Mr Alan Whitrow, the woodyard manager, told me that the bunker sensors never worked, in the sense that they were never accurate. It was much easier to check the levels visually, which was precisely the reason for operators and managers doing so on a regular basis. Mr Whitrow did not agree with Mr Callaghan's assessment as to general practice in relation to levels within the bunkers: he said that each week was different, and that it was difficult to say.

46. At the Claimants' request, Mr Michael Kent QC for the Defendant called Mr Brian Hayes, the company secretary of the Defendant since December 2012. At the time of these events, he had been chief financial officer. He was responsible for signing and declaring the truth of the Defendant's disclosure statements in this litigation. Mr Hayes was a somewhat cavalier witness who appeared to take the view that because the Defendant had disclosed information relating to the contents of the bunkers, viz. the Pybis data, that was the end of the matter. I was not impressed by that attitude, or by certain features of his evidence. It is unclear why no search for any electronic documents was made before the Standard Disclosure Statement was signed on 28th March 2013. In relation to the first Supplemental Disclosure Statement dated 25th June 2014, Mr Hayes had ticked the box to the effect that no search had been made for electronic data, but then it transpired that electronic meteorological data had been disclosed. Mr Hayes told me that he had made a mistake about this: that, in my view, evinced at best poor attention to detail. The further Disclosure Statement dated 14th August 2014, again signed by Mr Hayes, stated that a search had been made of the internal server in Portugal. On 17th June 2014 HHJ Gore QC had been told that a search would be made of that server, and that it would take 4 months.

47. The position in relation to the Sonae computer system was investigated with Mr Hayes, but his evidence was not as clear as it might have been. He told me that the SCADA data with which this case is concerned would not have been backed up or saved on Sonae's main server in Maia, Portugal. Those data were retained on temporary log files which were deleted after 4 weeks. My understanding of computer systems is imperfect, but I imagine that an expert would have been able to retrieve these data from the systems in Liverpool notwithstanding their deletion. However, on 2nd November 2012 the Sonae plant was sold to a competitor, along with its computers. Some data would have been transferred to Portugal for financial and other obvious reasons, but even if they still existed I see no reason for any of the SCADA data being preserved in this way: their value was ephemeral.

48. It follows that unless I reject Mr Hayes' evidence about the SCADA data not being transferred to Portugal at any stage, the data were deleted from the system within 4 weeks (Mr Hayes' figure) or 3 months (Mr Callaghan's), could not easily have been

excavated from the hard drive after that, and were lost forever on 2nd November 2012. I have asked myself why Mr Hayes took the trouble to make any lengthy inquiries in Portugal if the true position was that the data of interest did not exist by then. Upon reflection, and considering Mr Hayes' evidence as a whole, I cannot conclude that he was a mendacious witness. It was necessary to undertake certain inquiries in Portugal as to how the SCADA system functioned in relation to the bunkers, and the operation of Sonae's computer systems in general. Mr Hayes was an unsatisfactory witness, but I do not reject his evidence wholesale.

49. Mr Redfern also relied on the fact that in June 2011 Dr Jowett had advised the Defendant by email to retain electronic data relating to the firefly system, from which it should have been deduced that there was a need to retain electronic documents generally. However, I do not draw that inference at all: Dr Jowett was not interested in or impliedly referring to bunker sensor data, and in June 2011 the Defendant could not have anticipated these claims, or that the levels in the bunkers would be an issue.

50. At this stage, it is convenient to address Mr Redfern's submission that I should draw inferences adverse to the Defendant regarding the contents of the bunkers because it has lost or withheld relevant information, namely the records from the SCADA system relating to the data sent back by the sensors. He relied on the well-known case of Armory v Delamerie [1722] 1 Strange 505 which has recently been considered by Mann J in Gulati and others v MGN Ltd [2015] 1482 (Ch). In that case Mann J was considering breach of privacy claims by victims of phone-hacking. He decided that the Armory principle was relevant to his judicial task because on the facts of the case before him it was germane to the scope of the Defendant's wrongdoing, which was itself relevant to the scope of the invasion of privacy, and concomitantly relevant to damages [91]. Further, at [96] Mann J explained that Armory encapsulated an evidential principle relating to how the court should assess and find facts when that process has been obstructed by the acts of one of the parties.

51. Mr Redfern submitted that the Defendant, both through Mr Hayes and more generally, has acted reprehensively in failing to preserve the SCADA data before deletion, and/or failed to interrogate the main server in Portugal. On that footing he invited me to draw the inference that the bunkers were in fact at maximum operational capacity, which realistically (from the Claimants' perspective) means 3,000 Te.

52. Persuasively though they were presented, I cannot accept Mr Redfern's submissions. There are two answers to them. First, I do not accept that the Defendant should have preserved the SCADA data before the temporary log files were deleted, and/or before November 2012. Assuming that the Defendant's solicitors advised its client to retain relevant documents, including data, when this claim was first intimated, the Defendant's computer system in the UK was sold at an early stage in the litigation. It would have required considerable knowledge and "joined up thinking" to have concluded that these systems should have been interrogated, and temporary log files reconstructed, before they were transferred to the purchaser. By the time the Defendant should reasonably have been alert to this recondite issue, it was too late. Overall, the Claimants have fallen short of demonstrating the sort of reprehensible conduct on the Defendant's side which would justify the drawing of the adverse inference sought; and, in any event, the requisite causative link between conduct and the loss of electronic documents has not been established.

53. Secondly, the SCADA data, even if available, would have thrown little further light on the actual contents of the bunkers. The computer may have generated a percentage figure from the sensor data but it was, at best, only indicative. The clear evidence from Messrs Whitrow and Callaghan, which I accept, is that these data were not relied on. The only reliable means of establishing the bunker levels, and that was imprecise too, was to take a look. Operators and managers would not have taken the trouble to do this if their efforts were supererogatory. Furthermore, looking again at the diagram at paragraph 12 above, it is quite obvious that the inferences to be drawn from a series of distances from sensor to individual peaks were likely to be imprecise and inaccurate, in relation to a process which was dynamic. In the end, Mr Redfern had to submit that I should "draw an inference from an imponderable". The philosophers might enjoy this sort of metaphysical conundrum, but it is lost on the common lawyer.

54. Mr Redfern advanced a separate submission regarding the Defendant's comportment in relation to its disclosure obligations generally. This was less persuasive, given my judgment at the pre-trial review on 5th May 2015. Mr Redfern did not make sustained submissions about the shift managers' logs, and unless the Defendant is wholly misleading the court, the position must be that they are no longer available. I do accept, however, that the Defendant – through Mr Hayes, is remiss in failing to include these logs, as well as the temporary log files in its August 2014 Disclosure Statement, under the rubric of documents no longer in existence.

55. It is also convenient to deal with Mr Redfern's yet broader point, advanced more in cross-examination than at the end of the trial, that I should draw general inferences adverse to the Defendant flowing from its poor safety record, the number of fires, the inherent risk and the dryness of the work environment. Mr Redfern submitted that this fire was reasonably foreseeable, and that issues of foreseeability and causation are, by their nature, intrinsically intertwined. I cannot accept this submission either. The Defendant does not dispute that the Claimants' alleged losses are reasonably foreseeable. I do not accept that foreseeability and causation can be elided, or (to put the point slightly differently) that the degree of the Defendant's fault is capable of being relevant to the issue of causation. These are, and remain, discrete concepts. However badly the Defendant may have behaved, the Claimants still have to prove their cases to the requisite standard, and their task is by no means attenuated or abbreviated by the extent or quantum of breach of duty. The instant case is wholly different from the situation analysed by Mann J in Gulati (at [91] of his judgment) where the scope of wrongdoing bore on the quantum of damages, and also differs from the context of Viscount Simonds' dictum in Nicholson v Atlas Steel Foundry & Engineering Co. Ltd [1957] 1 WLR 613, 618 (once breach is admitted, it requires "little further to establish a causal link").

56. Mr Redfern advanced a separate submission on the drawing of adverse inferences based on a case extremely familiar to him, Wisniewski v Central Manchester HA [1998] PIQR 324, 340. He submitted that I should draw inferences adverse to the Defendant flowing from its failure to call relevant witnesses, namely Mr Sharkey and Mr Mitchell. I cannot conclude that these men could have given me *no* relevant evidence, but I do conclude that their evidence could not have assisted me significantly. Mr Mitchell could have spoken to the capacity of the bunkers, but this issue is no longer in dispute, *pace* Dr Mitcheson's arithmetical error in this second

report. As I have already said, Mr Sharkey could not improve on Mr Pybis' evidence, particularly in circumstances where the shift managers' logs are no longer available.

57. Returning to the quantity of woodchips in the bunkers, the final point which places the seal on the Defendant's case pertains to the separate calculations Dr Mitcheson performed to ascertain the approximate tonnage within the bunkers at the end of second stage of the fire. Paragraph 64 of Dr Mitcheson's first report dated January 2014 proceeded on the basis of an initial tonnage of 1,550 and a heat release rate of 330 MW, leading to a burn of 700 Te and a residue of 850 Te. In his report dated 12th September 2014, Dr Mitcheson carried out a more straightforward calculation based on the assumption that the bunkers were filled with unburnt woodchips up to the level of the letter boxes. On this premise, the mass of the unburnt woodchips was in the region of 900 Te, a figure not far off the 850 Te attained by a different methodology altogether. Mr Redfern sought to persuade me through cross-examination of Dr Mitcheson that the second method was either self-evident or tautological, amounting in effect to the skinning of the same cat by the same knife (my metaphor, not his), but I cannot agree. Somewhat rough-and-ready though Dr Mitcheson might have been in his later report, I consider that his methodology was different and the point he was seeking to make is valid.

58. Furthermore, Dr Mitcheson's advice to the court is that if there had been 3,000 Te in the six bunkers, then the heat release rate during stage 2 of the fire would have been a staggeringly high 1,000 MW. I accept his evidence that the degree of heat damage to the woodyard building tells against this having been the case.

59. It was put to Dr Mitcheson that an inference could be drawn from the fact that the similar quantities of ash seen in the bunkers after the woodchips had burned indicated that there were similar quantities *ex ante*. Dr Mitcheson's answers, which I accept, were that there was in fact less debris in bunker no. 2; and, in any event, that post-fire ash levels cannot give a reliable indication of the pre-fire load.

60. Overall, I am satisfied on all the evidence that the total quantity of woodchips in the bunkers was in the region of 1,550 Te. It might have been slightly more, it might have been slightly less, but it is reasonable to proceed on the basis of that figure.

The Remaining Fire Load

61. Dr Mitcheson has calculated the remaining fire load in the woodyard building. In his estimation, there was approximately 20 Te of conveyor belting, 16 Te of foam insulation and 4.5 Te (or 5,000 litres) of hydraulic oil. The combustion of these materials created dense black smoke. In addition, there were unquantified accumulations of woodchip and associated dust outside the bunkers which would not have materially contributed to the fire load but would have accelerated the spread of the conflagration at or shortly before 04:00 on 10th June. Finally, Dr Mitcheson refers to electric cabling with small amounts of plastic insulation, hydraulic hoses, miscellaneous painted surfaces and other similar combustible materials. Again, these did not contribute materially to the overall fire load.

62. Dr Mitcheson has taken into account the conveyor belting and the hydraulic oil in computing the heat release rate for stage 2 of the fire. In that regard, he has not taken into account the foam insulation (more exactly, the polyurethane foam filling the gap between the perimeter walls of the building), on the basis that it pyrolised and absorbed heat to more or less the same extent as it emitted heat. I do not understand that part of Dr Mitcheson's evidence to have been contradicted. In any event, the Claimants would not be assisted by greater heat release rates.

63. The Claimants dispute the quantity of foam insulation and contend for a figure of approximately 40 Te. They also argue that 4-5 Te of perspex roof sheeting was involved in the fire. Dr Mitcheson agreed that the total potential fire load as regards the foam insulation was in the region of 40 Te, and on one interpretation of his evidence in cross-examination he appeared to accept that had it burned it would have added considerably to the heat release rate. It is unclear to me why he made that concession (if indeed he made it) – logically, combustion should have a neutral impact on the heat release rate regardless of the quantity burnt – but his real point was that on his interpretation of the photographic evidence no more than 16 Te could have been consumed in the fire. This was on the footing that only the top 2m burned. Dr Jowett, who was in a better position to assess this damage, said in cross-examination that *"just the top few metres were involved in the fire"*. However, he also said that he had not done the calculation. In my judgment, it is likely that more than 16 Te of foam insulation was involved in the fire, but it is unclear how much more. I do not accept that the whole potential fire load of 40 Te was entailed. My finding has no impact on Dr Mitcheson's heat release rate calculation for stage 2, but it does serve to enhance the emissions of pollutants during that stage. Yet, how much difference that made cannot be assessed, save in the general sense that the enhancement could not have been that great in proportion to the quantity of woodchips. Finally, I cannot accept Mr Redfern's criticism of Dr Mitcheson that he should have accounted for the foam insulation. This expert's brief was to evaluate heat release rates; he had no interest in emissions and pollutants.

64. As for the perspex sheeting in the roof, it is clear that Dr Mitcheson did not take these into account. They would have had a minimal impact on heat release rates but would have contributed to some extent to the overall fugitive escape of potentially toxic chemicals. This is extremely difficult to quantify, not least because the dimensions of the roof spaces have never been measured, and to the extent that they appear in photographs their size and weight cannot be readily estimated. Mr Callaghan said that there were 3-4 pitches in the roof, with a maximum of 2-3 thin perspex panels in each pitch. With reference to furniture in the court room which Mr Callaghan used as his yardstick, I formed the impression that each panel was about 2m x 1.5m. I would not wish to speculate as to the total weight of all the panels, but the Claimants' estimate of 4-5 Te seems excessive. The panels melted at the outset of stage 2 and added to the mélange of chemicals at that juncture.

The Transition from Stage 2 to Stage 3

65. Stage 2 of the fire was characterised by very considerable quantities of thick, black smoke billowing forth from the roof of the woodyard building. There are photographs and spectacular video footage which illustrate this quite well, allowances being made

for photograph quality, the play of light sources across the lenses, and (at least in some cases) some doubt as to the exact timing of certain, publicly available video footage. The black smoke was the result of an oxygen-deprived fire, burning rich, producing incomplete products of combustion. Dr Mitcheson agreed in cross-examination that the space both immediately above and within the bunkers was heavily smoke-logged during stage 2, and that the richer the burn, the greater the pollutant content. Dr Jowett was asked about smoke-logging, and in his view what mattered was not the clogging smoke above the bunkers but within them. In my judgment, nothing really turns on this nuance.

66. In Dr Mitcheson's opinion, the transition from stage 2 to stage 3 occurred at approximately 10:00 on Friday 10th June, although he accepted that placing an exact timing on what was essentially a process rather than an event was somewhat arbitrary. Dr Mitcheson based his opinion on the MFRS log and what he considered to be reliable lay evidence (appended to his first report), and on the photographic and video record. I have reviewed the log very carefully but I am far from convinced that it provides much positive support for Dr Mitcheson's argument. Certainly by later on that afternoon there is reasonably clear evidence of a significant reduction in the overall quantities of smoke, and the Claimants propose a later time for the transition, namely approximately 14:30 on Friday 10th June.

67. There are two features of the evidence which require closer examination. First, what may be described as the highlights of the photographic and video evidence were considered by Dr Mitcheson, both in chief and in cross-examination. I should add that when opening his case to me Mr Redfern presented a 40 minute montage of the available evidence, and Dr Mitcheson confirmed that he had seen it. A video available on Youtube, which is untimed but Dr Mitcheson surmises was taken at around 10:00, shows lesser quantities of smoke issuing from the plant, and a possible change in colour – blending from white grey to a darker shade of grey. Photographic images taken at 10:28 and 10:29 that morning, showing the west and northwest aspects of the woodyard building, appear to depict smoke of a light grey colour tinged with yellow. There was some suggestion that the yellow might be a refraction or reflection of light from the sun, but I doubt that. Dr Mitcheson agreed in cross-examination that the yellow smoke was probably the product of something other than wood. However, it remains unclear what this was or might have been, and ultimately smoke of this hue does not really assist me in timing the transition from stage 2 to stage 3. Another image timed at 11:04, but taken from a different angle, shows what I would describe as grey, but not black, smoke. A further photograph taken at 11:17 shows a significantly reduced amount of smoke.

68. Dr Mitcheson agreed that the position changed at approximately 13:24 on the Friday afternoon when there is a body of photographic and video footage showing thick black smoke. By 14:49 the quantity had reduced but the colour was still fairly black. Dr Mitcheson's contention is that this represents the combustion of approximately 2,500 litres or 2.25 Te of hydraulic oil which had entered the immediate zone of the fire. There is some support for this explanation from a MFRS log entry timed at 13:29 – it erroneously refers to "approximately 1,500 litres of hydraulic fuel" having entered the area of the fire, but nothing turns on this quantitative error, or the mis-description of the substance.

69. On the other hand, the Claimants contend with some conviction that it is not easy to understand how 2,500 litres of hydraulic oil could have accounted for densely copious quantities of black smoke which endured for at least 85 minutes. Interestingly, Mr Callaghan put the duration of this black smoke as being only 10-15 minutes, which in my view is incorrect. I see the common sense force of the Claimants' point, but Dr Mitcheson explained in cross-examination that the oil could have pooled on the ground. Dr Mitcheson did not accept the force of the point that one would have expected both oil tanks to enter the fire zone more or less simultaneously. In this regard he received some support from the evidence of Mark Callaghan to the effect that the oil tanks were 8-10 metres apart.

70. To my mind, there are two difficulties with the Claimants' argument hereabouts. First, they have no expert evidence with which to contradict Dr Mitcheson, and overall I have concluded that he was a good witness. Secondly, if this was not hydraulic oil burning, then the only competing hypothesis is that there was some unexplained recrudescence in the fire involving the woodchips, and what might be characterised as a temporary reversion from stage 3 back to stage 2. Although I fully accept the possibility that this fire waxed and waned to some extent, I cannot agree that a flare-up of this magnitude might have occurred. All six bunkers were heavily consumed in the fire by 06:00, and I cannot accept Mr Redfern's subsidiary point that more polyurethane foam might have been involved after 13:30.

71. For all these reasons, I have concluded on the balance of probabilities that the transition from stage 2 to stage 3 occurred at approximately 10:00 on Friday 10th June, and that at that point Dr Mitcheson is correct in advising the court that around 700 Te of woodchips were consumed over the six hour period from 04:00 to 10:00, leaving approximately 850 Te at the point of transition. Dr Mitcheson derived this figure from the level of woodchips in relation to the "letter box" or slot openings. In my view, this was a somewhat rough-and-ready approach, but Mr Redfern did not take issue with it.

72. Nor did Mr Redfern take issue with another calculation which appears at paragraph 69 of Dr Mitcheson's first report. There, Dr Mitcheson explains that by 13th June the MFRS photographs show that the bunkers contained ash and smouldering woodchips to a depth equivalent to about three-quarters of each slot height, i.e. about 0.75 metres. This equates to a volume of about 225m³ in each bunker, suggesting an unburnt mass of about 22 Te per bunker and a total mass of about 120 Te. Accordingly, the majority of the woodchips remaining at the commencement of stage 3 were consumed by about midday on 13th June. Subsequently, it is obvious that there was much less left to burn, and Dr Mitcheson calculates that by 7th July only about 10 Te remained. Put another way, by midday on 13th June there was much less left to burn, much less smoke, and many fewer emissions of pollutants.

73. In my judgment, the Claimants cannot escape from the logic of these computations, nor did they try to. Dr Mitcheson was cross-examined on the basis that if the figure of 1,550 Te which he was given by Dr Jowett was wrong, then his report was wrong overall. As it happens, the figure he was given was – on my findings - correct, but even if it had been erroneous Dr Mitcheson would simply have had to revise his calculations on the basis of my finding; he would not have had to revisit his methodology.

74. Dr Mitcheson's calculations receive further support in the lay evidence from the Defendant's side. I was not particularly impressed by the evidence of Ms Joanne Ashton and Ms Gina Fitzgibbon – there were too partisan for my liking, and overly disposed to downplay the extent of the fire on the Friday morning. The evidence of Mr Alan Whitrow was very much more compelling. According to paragraphs 39 and 40 of his witness statement:

> "I returned to the site on Saturday 11th and Sunday 12th June 2011. During these days, I walked around and liaised with demolition contractors and the fire brigade. On a number of occasions I was inside the woodyard building. I was able to see clearly and did not experience any breathing difficulties. The fire fighters were not wearing any breathing apparatus as we walked around the site or were inside the woodyard building …
>
> During the remaining period of the fire, I continued to be on site during my normal working hours. By approximately Monday, the smoke from the fire had decreased significantly and from then onwards, the smoke gradually decreased."

75. Best of all from the Defendant's perspective was the evidence of Dr Jowett. Back in June 2011 he had no interest in matters such as the quantity, thickness and colour of the smoke; these were wholly outside the ambit of his concerns. As I have said, he went to the plant on 14th June in order to undertake a preliminary inspection. His photographs taken at the time do not show copious quantities of smoke, and when he inspected the woodyard building that afternoon there was very little smoke at ground floor level. The position was the same the following day. Dr Jowett returned to the site on 23rd June and carried out a risk assessment for his own purposes. This assessment made provision for wearing a personal gas monitor should the need arise, but according to paragraph 22 of his witness statement the alarm levels on the monitor were triggered at no point during his inspection. On that occasion, Dr Jowett took what he called an "inspection movie" (his exhibit 'PAJ-5', lasting 40 minutes) which shows little smoulder or smoke emanating from the bunkers by that stage.

76. Finally in this regard, there is an illuminating photograph published in a national newspaper showing the scene at 17:21 on 14th June. From the woodyard building emerge relatively modest quantities of grey-white smoke. The wind direction is towards the photographer. And between the photographer and the plant we see a mother and child, apparently oblivious to the smoke or its immediate consequences. True, one possible inference is that the woman in question was acting in reckless disregard of her own and her child's health. My preferred inference, having considered all the available evidence, is that the smoke was not causing any obvious, immediate ill-health effects.

77. Of course, the weight to be given to this corpus of evidence should not be exaggerated. It is far from being quantitative; it merely creates a general impression. On the other hand, it needs to be recognised that aspects of the Claimants' case depended on the making of a favourable impression from their perspective. That aside, I appreciate that from a more scientific viewpoint greater emphasis should be placed on considerations such as the applicable emission factors, the meteorological

variables and health thresholds to be drawn from the toxicology, all being matters which remain to be addressed in this judgment.

Heat Release Rates

78. The only evidence available to assist me on this issue came from Dr Mitcheson. Apart from a major attack on Dr Mitcheson's factual premises (e.g. initial tonnage of woodchips in the bunkers; amount of foam insulation), and a strand of cross-examination intended to reinforce his case in relation to emission factors, Mr Redfern did little to impugn Dr Mitcheson's methodology in this somewhat technical and recondite domain. In these circumstances, I am able to be quite brief.

79. As its name might suggest, the heat release rate is the heat generated by a fire measured in watts. In a case such as the present where any empirical evaluation would have been close to impossible, heat release rates can be computed from the product of the mass burning rate (measured in m^1 kg/s) and the heat of consumption, ΔH, expressed in MJ/kg.

80. Dr Mitcheson has computed heat release rates for the various stages of the fire, as follows:

- Stage 1, from about 17:30 on 9th June to about 03:35 on 10th June: $q^1 = 0.01 \times t^2$ MW (t being measured in hours).
- Stage 2, from about 04:00 to 10:00 on 10th June: q^1 = 330 MW (for the woodchips), + 28 MW between 04:00 and 06:00 (for the conveyor belts) + 12.5 MW between 06:00 and 07:00 (for the hydraulic oil).
- Stage 3A, from about 10:00 on 10th June to about 12:00 on 13th June: $q^1 = 64 \times \exp(-0.65 \times t)$ MW (where t is the time in days after reverse smouldering became predominant, on 10th June, at which time t = 0).
- Stage 3B, from about 12:00 on 13th June to 7th July: $q^1 = 1.4 \times \exp\{-0.1 \times (t-3)\}$ MW (where t is the time in days after reverse smouldering became predominant, on 10th June, at which time t = 0).

81. In reaching these conclusions, Dr Mitcheson made a number of assumptions about the nature of the fire at various stages, its levels of oxygenation, the calorific value of wood, its moisture content, and other matters. These were necessarily imprecise, and the resultant heat release rates cannot be regarded as entirely robust. Given the absence of an expert instructed by the Claimants who might have advanced different heat release rates, following which there might have been room for an element of compromise at a joint experts' meeting, at the conclusion of his evidence I invited Dr Mitcheson to reconsider his conclusions and advise me as to whether there might be respects in which a different opinion might be accommodated. Dr Mitcheson declined my invitation.

82. On my understanding of his cross-examination, Mr Redfern sought to undermine one of the assumptions made by Dr Mitcheson, namely that "*the combustion of the*

woodchips in the bunkers would be expected to resemble that of an under-ventilated, close packed crib within an enclosure" (see paragraph 63 of his first report). Shortly after lunch on the day Dr Mitcheson was giving evidence (Friday 5th June), Mr Redfern cross-examined him at length on the quantity of oxygen likely to reach the fire on account of the degree of smoke-logging within and above the bunkers. Mr Redfern's objective was to ensnare the witness into a debate about emission factors, not heat release rates, but Dr Mitcheson was unwilling to express a view about these. I suspect that he might have been qualified to do so, but it was clear that he had performed neither the calculations nor the degree of deep cogitation required to express a properly tutored view. Mr Redfern made some headway on this topic, in the sense that he was able to persuade me that certainly during the earlier hours of stage 2 there were very considerable quantities of black smoke and this fire was severely under-ventilated. However, I need to store this evidence away for use at a slightly later stage in this judgment, under the rubric of emission factors.

83. Although Mr Redfern was far more concerned to develop his point in relation to emission factors than to heat release rates, I pressed Dr Mitcheson on the issue at the very end of his evidence. Given that Dr Mitcheson had derived his mass burning rate set out at paragraph 63 of his first report from the SFPE Handbook and a situation which is there described as apt for a *"room fire with one or more vertical openings"*, I was concerned whether the particular features of this fire might displace or undermine this model. Dr Mitcheson had agreed in cross-examination that the simile was not exact, but he told me that he had taken proper account of the ventilation of this fire in arriving at his final heat release rate for stage 2. When I examine Dr Mitcheson's oral evidence against paragraph 64 of his report, I am not wholly persuaded that his analysis is correct. In particular:

> "The mid-heights of the slots were about 0.5m above the base of the bunkers and the openings measured approximately 25m x 1m each side, giving an area of 50m² per bunker. On this basis, the mass consumption rate is predicted to have been about 4.25 kg/s per bunker. However, this ignores the open top through which air would have entered and products would have escaped, both of which would increase the burning rate. For this exercise I have estimated that the burning rate during stage 2 would have been about 6 kg/s per bunker, or 33 kg/s overall."

84. No one has spotted the arithmetical, or more likely typographical, error in the final line of the foregoing quotation, and I have not redone the heat release rate calculation to fathom this issue. Given the ubiquity of dense black smoke and consequent under-ventilation of the fire during certainly the early hours of stage 2, I suspect that Dr Mitcheson has increased the burning rate by too high a factor in all the circumstances of this case. A slightly lower burning rate, and a concomitantly depressed heat release rate, might make a relatively small difference to the plume modelling, no doubt in the Claimants' favour – the higher the heat release rate, the higher the apex and range of dispersal of the plume. However, Dr Mitcheson's evidence has not been contradicted, it is not obviously illogical or implausible, and I have simply no evidential basis on which to reduce his estimated burning rate during stage 2.

85. A separate issue arises as to whether a deduction should be made for loss through heat radiation outwards from the smoke plume. Dr David Carruthers, the Claimants'

modelling expert, assumed that 30% of the convective heat would have been lost through radiation. In his evidence in chief, he explained that it was *"pretty standard in fire models to account for loss due to radiation - it is heat lost through the buoyancy of the plume"*. I appreciate that this is Dr Carruthers' standard practice, but his expertise in this area is questionable. Although he has a doctorate in atmospheric physics, he is not a fire engineer. Even so, I am prepared to accept that Dr Carruthers is qualified to speak in broad terms about the natural propensity of a hot body to lose heat to a cooler surface. That is basic physics.

86. Mrs Angela Spanton of Envirobods did not make any allowance for heat loss through radiation. Her evidence on this topic was not altogether clear, but she appeared to be saying that the ADMS fire document takes radiative loss into account, the radiative heat would have been retained or entrained within the smoke, and in any event *"the smoke would absorb some heat, but not 30%"*. I fail to understand her reference to absorption of heat within the smoke: the issue is whether heat would have radiated away from the smoke plume. Mr David Shillito, the Defendant's chemical engineer, was qualified to comment on this issue, but was not particularly helpful in giving me a figure upon which I could proceed. He refused to accept that 30% might be lost through radiation; he accepted that some might be dissipated that way, but he would not quantify it.

87. I am critical of the Claimants for not cross-examining Dr Mitcheson on this issue. He was the expert best placed to deal with it, and it is clear from paragraph 24 of his first report that he had factors such as *"incident radiant heat flux"* in mind. What is entirely unclear is whether Dr Mitcheson made any allowance for this in his computations, although I believe not because there is no further mention of the issue. Doing the best I can in these unsatisfactory circumstances, I am prepared to accept the probability that some heat would have been lost through radiation, but I am completely disinclined to embrace Dr Carruthers' 30%, which is based on little more than assertion. My deduction has to be somewhat intuitive and less than wholly evidence-based, but in my judgment, applying the probabilistic standard of proof, the correct deduction in this case is one of 10%.

88. It follows that I am accepting Dr Mitcheson's methodology and computations in relation to heat release rates (see paragraph 80 above), and am deducting 10% across the board for radiative heat loss. Overall, Dr Mitcheson was a good witness, although the chemistry between him and Mr Redfern was at times antagonistic. No blame for this slightly combustible situation needs to be apportioned. It would have been preferable had Dr Mitcheson been required to grapple during the forensic process with an expert in like discipline called by the Claimants, but the state of affairs which obtained has absolutely nothing to do with him.

Emission Factors

89. This was the most hotly contested scientific issue in this case, and by far the most difficult. The expert antagonists were Dr David Carruthers for the Claimants and Mr David Shillito for the Defendant. Dr Carruthers was involved in the development of the ADMS plume model which both parties' experts have used to provide the court with an indication of the locations and wanderings of the smoke plume over the

relevant period. I have already alluded to the fact that he is not an engineer, and emission factors fall within the discipline of chemical engineering. Mr David Shillito brings precisely that expertise to the forensic table. However, Mr Kent did not object to Dr Carruthers expressing opinions on this issue. He was quite right not to do so. Dr Carruthers' scientific background as a physicist, and his vast experience in the use of emission factors in numerous situations, enables him to speak authoritatively on the matter.

90. Dr Mitcheson and Mrs Spanton declined to express an opinion on the issue of emission factors. I have already observed that Dr Mitcheson was probably qualified to do so, but the topic did not fall within the ambit of his instructions. Mrs Spanton was less well-placed than Dr Carruthers to assist, and from her perspective she was doubtless well aware that the Defendant's nominated expert in this regard was Mr Shillito. I draw no inferences adverse to the Defendant from their silence.

91. Throughout the trial, expert witnesses used the terms "emission factors" and "emission rates" almost interchangeably. No confusion was created in their minds by this approach, but I felt that this terminological schizophrenia created the risk that the lawyers, including of course myself, were in danger of intermingling the concepts. Regrettably, it has taken me some time to fathom this issue.

92. In very general terms, the endeavour is to quantify the generation rate of any chemical or particle of interest over any given period of time. It is this metric, the emission rate properly so called, which is factored into the plume model. The smoke plume contained a cocktail or soup of chemicals and particles, and their individual emission factors varied. Beyond this general explanation, it is possible to be more precise. Strictly speaking, an emission *rate* is the product of the emission *factor* for any given chemical or particle and what Dr Carruthers called the "burning rate" (i.e. Dr Mitcheson's heat of consumption, not the same as his heat release rate) for the remaining fire load at the material time. Confusingly, Mrs Spanton's preferred terminology is not "burning rate" or "heat of consumption", but "heat output". In ordinary parlance, a *rate* is, of course, time related, whereas a *factor* a fraction. On this approach, an emission *factor* is measured in terms of the mass of the chemical or particle of interest per equivalent mass of the available combustible material (usually, g/kg); the burning *rate* (a.k.a. heat of consumption or heat output) is measured in MJ/kg (see paragraph 79 above); and the resultant product, the emission *rate*, is measured in g/s/MW.

93. Confusion arises because the experts have tended to use their terms interchangeably, confident in the knowledge that each fully understands the other, but possibly oblivious to a lawyer's very stale basic science. For the avoidance of all doubt, it may be helpful if I were to set out my understanding of the position. If the emission factor is 100 g/kg, and the burning rate or heat of consumption is 10 MJ/kg (as Dr Mitcheson says it is), 100 g/kg becomes 10 g/MJ. As a joule is a watt per second, this becomes 10 g/s/MW. As Envirobods have explained, there are other ways in which the same result may be attained, entailing the application of a relatively simple formula. Thus, what looks like a rate, because it is measured over time, is described as a factor.

94. Now that the position is fully understood, no confusion arises. However, it is necessary to be clear as to where this leads. Whatever the terminology, the plume

modellers have used two separate variables to arrive at the generation rate over the course of the fire of the chemicals and particles of interest. They appear to have used an emission rate expressed in mg/s/MW (although they have called it a factor), and they have also used an appropriate burning rate. This is the key concept Mr Redfern wanted me to understand. It is obvious that as the quantity of combustible material falls, so must do the generation of the relevant chemicals and particles. According to Dr Mitcheson's calculations, the heat release rate falls exponentially after midday on 13th June, and on my understanding what the plume modellers call the burning rate or heat output is logarithmic (assuming that my recollection of basic differential calculus has not altogether deserted me). In truth, it matters not, because the appropriate variable is factored into the ADMS model. However, it is important to keep separate the concepts of emission factors and burning rates, since otherwise there might be a tendency to conclude that because the fire is dying down the emission factors must also be falling commensurately. The emission *rate* will be falling, but not necessarily the emission *factors* – unless, that is, other considerations come into play which independently impact on the latter.

95. Armed with the foregoing scientific logic, Mr Redfern submitted that the emission factors for this fire must remain constant over stages 2 and 3. However, that in my view is overly simplistic an approach. Putting to one side the reduction in the burning rate, the parties' experts are in fact agreed that the emission factors vary as the fire progresses. This is a consequence of variables changing independently of the burning rate, including the chemical environment changing as well as the degree of ventilation and efficiency of combustion. The experts are, however, far apart on matters of degree.

96. The smoke plume contained hundreds if not thousands of chemicals and particles of potential interest. The expert reports range over a vast array of chemicals and toxins, but in the events which have occurred I may short-circuit some of the debate. The present case is concerned with chemicals which possess irritant qualities and with micro-particles. The possible relationship between the two will be discussed in my section on toxicology. The chemicals with irritant properties are primarily the aldehydes, but there were others too within the fire plume that have not been quantified in any meaningful way, whether in terms of their emission factors, the toxicology or the plume modelling. The parties agree that the most important of the aldehydes for present purposes is acrolein. Apart from the irritant chemicals, the parties have been focussing on particulate matter which is less than 10 micro-metres in diameter (the PM_{10}) and the further sub-set of particulate matter which is less than 2.5 micro-metres in diameter (the $PM_{2.5}$).

97. The parties are in agreement about the emission factors for acrolein in particular and the aldehydes in general. For the former, Mr Shillito has undertaken a literature review and arrived at what he describes as an emission factor of 114 mg/s/MW. For the reasons he gives in his first report, and which I accept, this is very generous to the Claimants. Adopting the same liberal approach yields an emission factor of 600 mg/s/MW for all the aldehydes. The experts are also in agreement that these emission factors may be deployed over the whole of stages 2 and 3 of the fire. For reasons which are fully explored in paragraphs 114-118 below, I disagree with their approach, but I have no evidential basis on which to advance different values, and (in the face of

expert agreement on the issue) it would not be right for me to do so. The Claimants do far better out of this state of affairs than does the Defendant.

98. Where the parties part company is in relation to the PM_{10}s and the $PM_{2.5}$s. In this respect, the PM_{10} emission factors are the most important, because the factors for the smaller micro-particles are a derivative of the larger. Here, I need to take time to explain the basis of the differences between Dr Carruthers and Mr Shillito.

99. Dr Carruthers has examined three sources in the literature. First, the publication entitled US EPA Emission Factors AP42 Compilation of Pollutant Emission Factors addresses a number of potential analogues to the present fire, and Dr Carruthers considers that the best comparable is the wood-burning residential fireplace. According to this source, the emission factor for PM_{10}s in lbs/tons is 34.6, which translates to 17.3 g/kg. Two sections of the narrative section of this publication are relevant:

> "1.9.2 Fireplace emissions, caused mainly incomplete combustion, include particulate matter, mainly PM_{10}s … significant quantities of unburnt combustibles are produced because fireplaces are inefficient combustion devices, with high uncontrolled excess air rates and without any sort of secondary combustion. The latter is especially important in wood burning because of its high volatile matter content, typically 80% by dry weight.
>
> …
>
> Fireplace emissions are highly variable and are a function of many wood characteristics and operating practices. In general, conditions which promote a fast burn rate and a higher flame intensity enhance secondary combustion and thereby lower emissions. Conversely, higher emissions will result from a slow burn rate and a lower flame intensity. Such generalisations apply particularly to the earlier stages of the burning cycle, when significant quantities of combustible volatile matter are being driven out of the wood. Later in the burning cycle, when all volatile matter has been driven out of the wood, the charcoal that remains burns with relatively few emissions."

100. In his final submissions Mr Kent drew attention to a further passage in the US EPA document relating to "wood residue in boilers". This indicates an emission factor equating to 3.1 g/kg. However, none of the experts was invited to comment on this.

101. Secondly, Dr Carruthers referred to a paper by Stec et al published in the Fire Safety Journal [44 (2009) 62-70], Comparison of Toxic Product Yields from Bench Scale to ISO Room. I am far from convinced that much may be derived from this paper. In Figure 6 of the paper, the authors are considering amongst other things the product yields (i.e. emission factors) for soot from the burning of MDF for a steady-state tube furnace, compared with an ISO room, as a function of the carbon dioxide/monoxide ratio. Thus, the emission factor for a different product, namely soot, is analysed in comparative terms in a context distant from that of the instant case. As a separate

matter, Mr Kent relied on Figure 6 in support of his argument that emission factors decrease significantly as the fuel load is consumed. I agree that this Figure does show that the emission factor for soot decreases exponentially as the carbon dioxide/monoxide ratio increases, but without a much better understanding of this paper than anyone was able to give me, I cannot accept that it necessarily supports the point Mr Kent was seeking to make. On the other hand, I do accept that this paper lends further support for the proposition, if such support were needed, that increasing the ventilation reduces the emission factors.

102. The centrepiece of the Claimants' case on this issue was the document co-authored by Larson and Koenig, A Summary of the Emissions Characterisation and Non-cancer Respiratory Effects of Wood Smoke, a version of which was also published in the Annual Review of Public Health in 1994. Table 1 of the Summary gives the PM_{10} emission factors for "conventional wood stove" and "conventional fireplace" at, respectively, 7-30 and 15-32 g/kg of wood. The narrative section of this paper reads, insofar as is material, as follows:

> "The large variability in emission rates for a given appliance is due to a number of factors including stove design, wood moisture content and burn rate … In conventional stoves, increasing burn rate increases combustion temperatures and efficiencies, but in catalytic and non-catalytic devices the higher burn rates actually decrease combustion efficiency by decreasing the times in the secondary combustion rate …
>
> Compared to wood stoves, we know little about fireplace emissions. Here we distinguish fireplaces from conventional fireplace inserts. Inserts are a home-heating device with emissions similar to conventional wood stoves. Standard open fireplaces can be a net home cooling device because of the large amounts of air they draw from outside during maximum burn rates. In general, conventional fireplaces emit comparable amounts of particulate matter and less carbon monoxide per kg wood burned compared to conventional wood stoves. However, fireplaces usually operate at higher wood burn rates and for shorter time periods than most wood heating devices."

103. On the basis of the US EPA Emission Factors, Dr Carruthers has taken in his March 2014 report an emission factor for PM_{10}s of 17.3 g/kg. Looking at the Larson and Koenig paper, this falls towards the middle of the range. For $PM_{2.5}$s Dr Carruthers has used a ratio of 0.82 derived on my understanding from the Californian Fires paper, addressed more fully below. No issue arises as to the ratio deployed, and the resultant emission factor for $PM_{2.5}$s is 14.2 g/kg.

104. Dr Carruthers prepared a document for the Joint Experts' meeting on 24th October 2014 (privilege has since been waived) which reduces these figures by 20% to reflect the moisture content in the woodchips. Envirobods did not take issue with that approach, although I note that Mr Shillito, who was advising them, did not make a similar deduction from his much lower figure. The point was not explored with any of the experts: either the parties assumed that it was not in issue, or it was overlooked. Towards the end of the trial, I indicated to the parties that I considered that the 20%

deduction was incorrect in principle. My understanding of the literature, including the US EPA document, was that "dry" wood did not mean wood which was 100% devoid of moisture, but wood that was at the drier end of the spectrum. I recognise that I was running solo with a point which had not appealed to Mr Redfern. At that stage the parties did not comment on my rationale or conclusion, but after the close of the proceedings and following his interpretation of an apparent concession by Mr Redfern in post-hearing email exchanges, Mr Kent returned to it and urged me to reconsider. His submission was that Dr Carruthers accepted the 20% deduction, and that it was supported by the US EPA's reference to "dry" wood in connection with the emission factors for the residential fireplace model.

105. I appreciate that I am responsible for setting up an issue which would not otherwise have arisen. However, Mr Kent had every opportunity to disabuse me of it before the trial ended. Further, I remain of the view that the 20% deduction is incorrect in principle, and the Claimants press me to adhere to that position. At paragraph 4.14 of his September 2014 report Mr Shillito, when addressing CERC's emission factors for the micro-particles, did not contend that they should in any event suffer a deduction for moisture. On its natural and ordinarily meaning, "dry" does not mean wood which has been notionally drained of all moisture. In any event, the US EPA document states that the moisture content of wood ranges from 5-70%, and that "dry" wood includes wood with anything up to 20% moisture. I do not read paragraph 1.9.2 of the US EPA review, with its reference to dry wood, as indicating that all moisture is squeezed out of the computation. Finally, I am not prepared to finesse this issue by allowing a deduction lower than 20%. It follows that, although moisture should obviously be taken into account in relation to the heat release rates (as it has been), it should not be in relation to emission factors.

106. Dr Carruthers advanced reasons of his own in justification of the US EPA residential fireplace emission factors, but before considering these it is convenient to set out Mr Shillito's reliance on different literature sources.

107. In Mr Shillito's view, the closest analogue to stage 3 of this fire is the wood-burning biomass boiler. His reasons in support of that thesis are summarised below, but his source is a Report to the Scottish Government dated September 2008, AEA Energy and Environment, Measurement and Modelling of Fine Particulate Emissions from Wood-Burning Biomass Boilers. Mr Shillito draws attention to Table 2.5 in that report, which summarises the findings of the Nussbaumer et al study (2008) on "typical PM emission factors" for four different boiler types including biomass boilers. Although the report refers to 80 g/GJ, this is equivalent to an emission factor of 0.8 g/kg. It may immediately be seen how far apart the experts are on this issue.

108. According to paragraph 2.1.1.4 of the report:

> "Smaller biomass boilers are generally not fitted with any pollution abatement devises as these are generally not required to meet current CAA requirements for emissions. However, most larger new automatic boilers are fitted with some form of flue gas cleaning device to remove particle (dust) from the flue gas before release to the atmosphere."

109. Mr Redfern quite rightly drew this passage to Mr Shillito's attention, and the latter agreed that larger biomass boilers operating in the UK would possess a flue gas cleaning device which would serve to reduce emission factors. On the other hand, it is clear from the report's analysis of the Nussbaumer at al study that the Austrian group which performed it were looking at a wide range of boilers, and included within scope "worse case emissions". When an examination is made of Table 2.6 in the Scottish report (which was not carried out during the trial), it is clear that the average PM_{10} emission factors derived from local monitoring data were markedly lower than those noted in Nussbaumer et al. It was perhaps for this reason that Mr Shillito felt able to state in this report that Table 2.5, on which he was relying, probably over-stated the position.

110. In elaboration of his position, Dr Carruthers explained that the fireplace analogue, albeit imperfect, represents the closest fit to the circumstances of the fire with which we are concerned. The basic principle, which is universally accepted, is that the better the ventilation the better the efficiency of the burn and the lower the emission factors for any chemical or particle of interest. There are other considerations which impact on emission factors, such as the chemical constituents of the fire load as the fire progresses, but these may be difficult to quantify. A residential fireplace is an open, uncontrolled fire which is reasonably well ventilated. Thus, in stage 2 of the fire this model would in Dr Carruthers' view (as articulated at one stage in his oral evidence) tend to underestimate the true emission factors for the micro-particles because it is accepted by Dr Mitcheson that the fire was under-ventilated. The fireplace model would be more apposite for stage 3. Given that Dr Carruthers has chosen to use one emission factor for the small particles throughout stage 2 and 3 of the fire, the Claimants submit that he has in fact under-estimated the position.

111. On the other hand, I agree with Mr Kent that Dr Carruthers' final position was that he accepted that he had used an emission factor throughout which was based on stage 2 when there was thick smoke with poor visibility. He said, *"I think we modelled the phase 2 fire. So we were looking at when the fire was clearly very thick with smoke"*. He also accepted that his modelling was on a worst case basis.

112. Dr Carruthers' basic objection to the wood-burning biomass boiler is that it is designed to achieve an automatic, controlled process, in other words an efficient burn, where the emission factors would inevitably be low. The presence of thick clouds of black smoke during stage 2 is completely inconsistent with the controlled process within Mr Shillito's presumed contemplation. Furthermore, a biomass boiler would have to be compliant with the Clean Air Act 1993, where stage 2 of the fire plainly was not. Dr Carruthers also sought to draw inferences from certain photographs relating to how far one could see through the smoke, but this evidence was too subjective and impressionistic for my palate.

113. In his oral evidence Mr Shillito elaborated on why he believed that the biomass boiler was a better analogue than the residential fireplace. In his evidence in chief he explained that (a) the fuel was very different, and he was looking for *"an effective fuel burnout"*, and (b) given the dimensions of our fire, it was appropriate to compare it to a model which was larger than a residential fireplace. Like Dr Carruthers, Mr Shillito in his reports had adopted a single emission factor for both stages of the fire, although had queried the methodological soundness of this approach. On my reading of his written evidence to the court, Mr Shillito was adopting an emission factor which he

considered was appropriate for stage 2, and then applied the same value to the beginning of stage 3. In his oral evidence, however, Mr Shillito said that his approach had always been to seek to ascertain the appropriate emission factor for stage 3a of the fire (i.e. 12th June), as he called it, and then apply the same value to stage 2. Mr Shillito struck me as an entirely honest witness, but I do not read his reports in that way. I consider that he was extrapolating from stage 2 to stage 3, not the other way round.

114. In his oral evidence, Mr Shillito conceded that his unitary emission factor of 0.8 g/kg was too low for the first two hours of stage 2 by a multiple of 3 or 4, but otherwise he adhered to his figure. Mr Redfern sought to exploit this concession by putting to Mr Shillito in cross-examination that, given that all aldehydes and other emissions are the result of incomplete combustion, logic would surely suggest that *all* Mr Shillito's emission factors should bear the same increment. Mr Shillito was surprised by this question, and did not really answer it satisfactorily, but the reality is that the premise on which the question was posed was completely incorrect (I must confess that the enthusiasm with which I harried Mr Shillito about this was misplaced). All emissions are the product of incomplete combustion (i.e. complete combustion of wood would produce carbon dioxide, water and nothing else): the issue is not the *fact* of emissions, but their relative quantities. Further, the parties have proceeded on the basis of agreed emission factors for all aldehydes and acrolein in particular – these are safely derived from the literature, and are not related to the emission factors for the micro-particles. In any event, multiplying the emission factors for the irritants by even a factor of four for the first two hours of stage 2 makes no material difference to the outcome of this case.

115. Mr Shillito recovered much of his poise during Mr Redfern's extended and able cross-examination of him on the issue of emission factors for the micro-particles. Mr Shillito rejected the fireplace analogue because in his view it predicates flaming combustion, which this fire was not. He stated that the key point in the instant case is that by the beginning of stage 3 the fire load was charred woodchips in respect of which the volatiles had already been released (during stage 2). Mr Redfern put to Mr Shillito the concept of "re-volatilisation", but this was simply not understood: the volatiles in Mr Shillito's view would burn off once and for all when initially released. As for the biomass boiler model, Mr Shillito accepted the point about statutory compliance, but stated that the analogue remained apposite because wood-burning boilers use a continuous source of woodchips (which, therefore, continue to emit volatiles and aerosols), and a larger combustion bed tends to be more efficient.

116. It was put to Mr Shillito that during stage 3 the fire was inefficient. He disagreed, and the transcript of the relevant section of his cross-examination reads as follows:

> "The fire was efficient in stage 3. It had plenty of oxygen in stage 3. My emission factors were designed for stage 3.
>
> Q. It was reverse smoulder. That's not efficient oxygen, is it?
>
> A. I think you will find it was quite efficient.
>
> Q. You can't say that stage 3 was an efficient burn, can you?

A. It's rather like a coke brazier.

Q. The fire is burning back on itself. That's reverse smouldering, isn't it? It's inefficient.

A. The oxidation is happening at the surface. The heat is penetrating downwards. As the top fuel burns away, the fuel below comes alight.

Q. It's only the top –

A. If you measure the carbon monoxide from a coke brazier, you will find there is some, but the burning combustion efficiency is quite effective. It's like the barbecue after the main thing is alight. It's a coke brazier."

117. Notwithstanding that his own expert had been content to deploy it, Mr Redfern ambitiously sought to persuade Mr Shillito that the residential fireplace model was inapposite in the sense that its application would tend grossly to under-estimate the emissions from the Sonae fire. Mr Redfern's point was that a fireplace has flame – which burns off emissions - whereas our fire did not. Mr Shillito did not accept that this was so:

> "The fire, I think, is -- if you start at the top of the page, that fireplaces are primarily used for aesthetic effects and secondarily as supplemental heating. The fire is designed to be pretty, to show a nice red flame. The red flames in themselves produce carbon black. The redness of the flame is in fact carbon black glowing in the flame. So the fire is designed to produce soot which burns in the flame. The volatiles, which contain the irritant substances, will burn preferentially in the flame to the carbon. So as I see it, a nice red flame will produce black soot, but probably low volatile contents, and that's one part of it. The volatile material escaping from the fire will depend obviously on the way that the fire is arranged. But to my mind, the black soot from the open fire is contributed by the loss of heat to the outside environment, reducing the -- this is radiation loss from the visible fire, and effectively quenching the red flames. This is the soot in the basic wood fire chimney."

118. My approach to this important issue is as follows. Unlike the experts, I am not prepared to adopt a unitary emission factor for the whole of stages 2 and 3. In my judgment, the emission factors changed significantly over the course of the fire, and to take either a rough-and-ready or an average value would not achieve justice. I will take one emission factor for stage 2 and another for stage 3. Each will be at or near the top of the range in both instances.

119. With respect to Mr Shillito, who I felt was a very fair and helpful witness, I cannot accept that the present case receives its best analogue from the wood-burning biomass

boiler. This is a controlled, automated device designed to minimise emission factors, and the instant case is concerned with an admittedly very large uncontrolled fire.

120. In the absence of experimental or empirical data collected at the fire scene, I consider that the residential fireplace model is the best that may be achieved in these circumstances. As the authors of the US EPA report make clear, a residential fireplace is an inefficient device and there will be significant variances between different homes, configurations and wood piles. The report also explains that emission factors will depend on - amongst other matters - burn rate, the degree of ventilation and the degree of flame intensity. Here, during stage 2 we have a fast burn rate, an under-ventilated fire (exactly how under-ventilated is not readily quantifiable) and low flame intensity. Two out of these three matters incline towards higher emission factors.

121. Dr Carruthers' value is derived from the US EPA report and he does not appear to have examined the Larson and Koenig report and paper which give higher figures for residential fireplaces. I do not accept Mr Kent's point that the Larson and Koenig data have in some way been superseded by the US EPA. The issue arises as to whether it is open to me to taken on board these data in all the circumstances of this case. Mr Kent objects to such a course, essentially on two grounds. First, he submits that there is no evidential basis for a higher value. Secondly, he submits that the Claimants should be bound by the figure Dr Carruthers has advanced on their behalf, and that it would be wrong and unjust to countenance a higher value at this late stage.

122. As for the first objection, I consider that there is an evidential basis for a higher value, namely the Larson and Koenig report. Mr Shillito was taken to it, and I cannot accept his reasons for rejecting the simile (see paragraphs 115-117 above) – at least as regards stage 2 of the fire. I am entitled to draw inferences from all the available evidence, including Mr Shillito's answers. Although common sense can be a dangerous guide in relation to scientific matters, I have reached the clear conclusion that for stage 2 the instant case must fall towards the upper end of the fireplace model, and of the spread of values proffered by Larson and Koenig. Stage 2 of the fire was characterised by voluminous quantities of dense black smoke engendered in a significantly under-ventilated environment. During its early stages, the "letterbox" slots were blocked. Moreover, Mr Shillito has not included the hydraulic oil, the foam and the conveyor belting in his emission factors for the micro-particles, nor has CERC modelled these (I understand that CERC has modelled the 0.8% contamination, using the parallel of household waste). Although these have not been quantified, the presence of these items (which on my findings were wholly consumed within stage 2) gives me further confidence in moving towards the upper end of the bracket.

123. However, it would not be right to go beyond the figure of 32 g/kg as the emission factor for the micro-particles, as at one stage Mr Redfern temptingly submitted I should. That would be a leap in the dark, without an evidential platform, and unfair to the Defendant. More precisely, whatever success he achieved in cross-examining Mr Shillito does not underpin, or warrant, so extravagant an approach.

124. As for Mr Kent's second submission that the Claimant should be bound by Dr Carruthers' value, I see the force of it but in the exceptional circumstances of the present case I am prepared to allow the Claimants an element of latitude. Given Mr Redfern's forensic success with Mr Shillito, it would not be in the interests of justice

or the overriding objective to hold the Claimants to the figure Dr Carruthers has put forward, no doubt in good faith but absent consideration of all available evidence. The Defendant is not prejudiced by this course. Furthermore, Mr Shillito was aware of Larson and Koenig because he genuflected towards them in his reports (although whether he was aware of the report, as opposed to the published paper, is less clear).

125. Taking all this evidence on board, I am required to alight on the correct value for the micro-particles during stage 2 of the fire. Doing the best I can on all the available evidence, my emission factor for the PM_{10} fraction is 27.5 g/kg. I reduce that by 0.82 for the $PM_{2.5}$s, i.e. to 22.6 g/kg. In reaching these figures, I should make clear that I have erred on the side of a liberal approach: in other words, these are generous to the Claimants, and are tethered to the start of stage 2, not the end.

126. The transition from stage 2 to stage 3 was not instantaneous, but in my judgment by a fairly early juncture within the longitudinal course of stage 3 the emission factors for the micro-particles had fallen significantly. As the authors of the US EPA study explain, later on in the burning cycle the volatiles burn out, charcoal remains, and the emission factors decline. This tends to chime with aspects of Mr Shillito's evidence. On the other hand, Dr Mitcheson proceeds on the basis that at the commencement of stage 3 there were still approximately 850 Te of woodchips left to burn. Although many of the surfaces of the individual woodchips were charred and most were "cooked", I do no accept that there was charring *tout court*. Accordingly, further volatilisation was a probable phenomenon. Yet, it is indisputable that the predominant characteristic of stage 3 was smoke which was between whitish and light grey in colour. Furthermore, I accept Mr Shillito's evidence that the fire during stage 3 had become more efficient in consequence of the enhanced ventilation - both through the slots and from above.

127. In my judgment, the emission factors for the micro-particles fell significantly during the course of stage 3. By how much is unclear. I do not propose to reflect this consideration in my figure, either by selecting more than one set of emission factors over the 27 days of stage 3, or by taking an average. Like Mr Shillito, I believe that the proper focus is on Sunday 12th June, when the wind was from the east. In reality, the graph of emission factors plotted against time would show a marked decline from mid-afternoon on Saturday 11th June (I take that point in time because the hydraulic oil had burnt out by then) and about midday on 13th June. Thereafter, the decline continued, but was very much slower. Fortunately for the calculations, Saturday 11th June is far less important for virtually everyone than the Sunday, because the wind was blowing from the west. Focusing therefore as I am on Sunday 12th June, my conclusion is that the emission factor for the PM_{10} fraction is 12.5 g/kg. Again, I reduce that by 0.82 for the $PM_{2.5}$s, i.e. to 10.25 g/kg. These factors apply to the whole of stage 3. As before, this represents a liberal approach, even though it is slightly below the Larson and Koenig bracket for the fireplace model. In this regard I have taken account of Mr Shillito's evidence regarding the efficiency of the stage 3 fire.

Ash and Dust Deposition

128. In his report dated 25th March 2015, Dr Mitcheson addressed the issue of ash and dust generation. In his opinion:

> "If ash and dust were produced and transported from the fireground, the most likely time would have been during the intense combustion that occurred during stage 2."

In other words, Dr Mitcheson rejects out of hand the possibility that ash and dust might have been generated during stage 3, and I understand his reasons for doing so.

129. As for stage 2, Dr Mitcheson has performed some straightforward calculations on the basis of a fairly limited literature base, and has concluded:

> "The larger volume of fly ash estimated above released during stage 2 of the fire would therefore have formed an average covering over this area of less than about 0.35 microns, i.e. about one third of one thousandth of one millimetre."

130. Dr Mitcheson was not cross-examined on this report. The plume modellers have not modelled dust and ash on the basis of any reliable data regarding the sequence and history of the fire (CERC's previous modelling has been superseded), and in scientific terms the issue may be taken no further.

131. However, there is a mass of evidence from the Test Claimants on this issue which needs to be considered. Given that I am not considering claims in nuisance which are not personal injury claims, this evidence is not directly germane to my fact-finding exercise. However, it does bear on the issue of credibility, and I will be returning to it later.

TOXICOLOGY

132. Even as regards relatively low exposures of short duration, the general principle of toxicology applies: "the dose determines the poison". However, at these low exposures in particular, considerable allowance must be made for human susceptibility and variability.

133. In this domain the forensic tournament was between Professor Alastair Hay for the Claimants and Mr David Shillito for the Defendant. It is immediately apparent that in one sense this was an unequal fight: Professor Hay's enormous lance pitted against Mr Shillito's smaller weapon. Professor Hay is Professor of Environmental Toxicology at the University of Leeds, and an international authority in his subject. He is extremely experienced and has sat on numerous governmental committees over the years. Mr Shillito is not a toxicologist at all; he is a chemical engineer. However, his experience in environmental issues, and his sense of fairness, cannot be doubted.

134. Despite this obvious mismatch, I had the feeling that Professor Hay was somewhat hampered by the instructions he had been given. His three reports are similar to one another and speak at quite a high level of generality and theory. They are not particularly quantitative. It is almost as if he was being discouraged from pinning his precise colours to the mast until as late as possible. Furthermore, I cannot place any weight on the following assertion in one passage in his March 2014 report, based as it

is on a misapprehension as to the extent of this fire, and on modelling evidence which has been superseded:

> "On the basis of what is known about the Sonae fire, the photographic evidence of smoke from the fire, modelling of the fallout of pollutants generated by the fire and the topography of the area, it is more probable than not that many thousands of people would have been affected by the pollutant emissions."

135. Fortunately, Professor Hay has been much more forthcoming in the witness box and in the second Joint Statement of these experts. I found him to be an extremely commanding expert witness. On a few occasions, he became slightly argumentative, donning the mantle of the advocate, but I understood his passion for this subject and the strength of his opinions.

136. The areas of controversy as between these experts have been significantly whittled down in the later stages of the forensic process.

137. Aside from the micro-particles which I will address below, and where there remains a dispute, the experts are in agreement that the focus must be on the irritant chemicals rather than on the asphyxiates. The latter will not have left the fireground in significant quantities. As regards the irritants, the focus has primarily been on acrolein, but consideration has been given also to the total aldehydes within the metaphorical soup of the fire plume. During the course of Professor Hay's evidence in chief, Mr Redfern invited his witness to perform from the witness box a calculation designed to estimate the additive effect of the agglomeration all the probable irritant chemicals in this mix, in other words to look further than the limited number of compounds previously considered. This calculation had not been attempted before, and I did not give the Claimants permission to adduce evidence of this nature in this unheralded fashion. I gave a short judgment at the time, and a transcript (for the parties' benefit only) appears on Livenote.

138. Professor Hay's evidence was that irritancy is an immediate, not a delayed, effect. Once the chemical of interest reaches a certain dose, the body will respond adversely and defensively. Professor Hay added that the effect is usually transient, depending of course on dose and duration of exposure. The position is clearly explained in Dr J.C. Wakefield's paper for the Health Protection Agency entitled A Toxicological Review of Products and Combustion (2010). At paragraph 3.2:

> "The injury following exposure to an irritant gas depends upon the chemical involved, its concentration, the exposure duration and its solubility. However, the initial effect of exposure to these irritant gases is likely to be sensory irritation. Irritation of the eyes will cause pain and stinging of the eyes, initiation of a blinking reflex and lacrimation … An additional characteristic sign of exposure to irritant gases is a burning sensation of the mucous membranes of the upper respiratory tract, including the nose, mouth and throat. Pulmonary irritation will commonly occur following sensory irritation, due to inhalation of the irritant gas into the lungs. The irritation of the lungs gives rise to bronchoconstriction, coughing and breathing difficulties."

139. Professor Hay stated in cross-examination that there is nothing in the literature linking dermatological problems to smoke exposure.

140. As for the level at which a human being may react adversely to an irritant, it seems to me that I should be primarily focusing not on workplace or AEGLs, but on short-term levels of a different nature. Here, the HPA review paper is of assistance. It recognises that acrolein is the most potent of the irritants, and various thresholds are given at page 15 of the document. In this regard it is necessary for our purposes to "translate" parts per million into micro-grams per cubic metre, and doing the best I can with the arithmetic one arrives at the following:

(i) eye irritation resulting from exposure to acrolein has been observed at concentrations *as low as* c.146 μg/m³ (my emphasis), nasal irritation at c.356 μg/m³, and respiratory symptoms at c.572 μg/m³ (I note that Mr Kent's arithmetic is slightly different). Like Mr Kent, I assume that these values must be for healthy subjects. There is no evidence as to what they might be for vulnerable individuals, and I cannot accept Mr Kent's thesis that healthy people will be more sensitive to an irritant.

(ii) concentrations of acrolein in the region of 1,100 μg/m³ to 11,000 μg/m³ have been shown to cause the onset of lacrimation and eye irritation in human beings within a 10-minute exposure period.

(iii) acrolein at 2,640 μg/m³ has been reported to cause lacrimation in human beings within 5 seconds of exposure.

141. On my understanding, these data are agreed between the experts, but they need to be interpreted. Item (i) above is addressing the absolute minimum levels at which human beings might begin to experience symptoms. Most healthy persons will not experience symptoms at such levels or anything like them (assuming these values are correct), but the higher the dose the more individuals will begin to suffer. Further, the paper does not throw any light on duration of exposure (and note item (iii)), although the experts appear to have interpreted it as a trigger value regardless of length of exposure. Taking a threshold of 146 μg/m³ for acrolein in the context of its potential to cause eye symptoms is extremely favourable to the Claimants. The plume modellers have used this value, at my request, for the purposes of illustration. I should emphasise, if further emphasis were required, that exceeding it does not mean that personal injury was caused or even likely caused.

142. For completeness, I should add that from page 16 of the HPA document it may be gathered that the 8-hour threshold value for acrolein is 237.5 μg/m³, being a weighted average over that period, and the 15-minute value is 712 μg/m³. I have already said that these are not my primary focus, but no Test Claimant comes anywhere close to these modelled levels of exposure.

143. In his plume modelling Dr Carruthers has used an odour threshold for acrolein of 0.38 μg/m³. Mr Shillito's explanation for this figure is that it represents the threshold at which 50% of the population will recognise the smell (but would not be able to name it as acrolein). On my understanding of his evidence, Professor Hay felt that this was far too low - he suggested something in the region of 460 μg/m³ - but this may have been a complaint threshold rather than an odour recognition threshold. For present

purposes I consider that it is helpful to proceed on the basis of the 0.38 μg/m³ value, not in any way as a health threshold (*pace* Dr Carruthers' view that it is a *"good surrogate as a sort of envelope of the fire impact"*), but as indicative of being the sort of level at which many people would have been able to smell the smoke plume.

144. Aside from acrolein, Mr Shillito has also advised the court as to an appropriate health threshold or trigger value for "total aldehydes", and has alighted on a value of 500 μg/m³. As I have already said, Professor Hay did not perform a similar exercise for the purposes of his reports, but when I asked him at the very end of his evidence about Mr Shillito's figure, he told me that his recent calculations were not "far off". Much later, I had a colloquy with Mr Kent about the significance of this value. I had found it difficult to understand why the threshold for "all aldehydes" was higher than that for acrolein. The supposition must be that there is some sort of dilution effect, which I can understand in an experimental setting where relative concentrations may be in play; but in the context of a given smoke plume with a number of chemicals of the aldehyde family, one would have thought that the act of placing more of these under the microscope would serve to increase, rather than reduce, the irritancy potential. Mr Kent did not really advance a persuasive rebuttal of this, but overall it matters not. There is no evidential basis, or evidence-based methodology, which might enable me to reduce the acrolein threshold *below* 146, 356 or 572 μg/m³ to reflect the aggregation of aldehydes.

145. The HPA report also contains helpful narrative exposition under the rubric "smoke behaviour" (page 35):

> "The exposure to individuals to the fire effluent in the zone outside the immediate fire zone (zone 2) would be expected to be of most concern to public health … The major immediate hazard to public health in zone 2 is therefore, expected to be exposure to irritants and particulates generated in the effluent. Low concentrations of the irritant gases may cause significant irritation of the eyes and respiratory tract, which may affect a large number of people who are not directly exposed to the fire plume. The adverse effects resulting from exposure to these irritants are likely to be completely resolved following removal from the exposure, with no long term sequelae. However, the generation of more complex products such as PAHs, dioxins, dibenzofurans and particulate matter are of concern, but are likely to present a significantly greater hazard from long term or repeated exposure than following a large single acute exposure."

146. Where these experts disagree is in relation to the micro-particles. Hereabouts, the disagreement resides on at least two levels.

147. The first issue concerns the nature and mechanism of any irritant effect of the micro-particles. On my understanding of his evidence, Mr Shillito does not consider that the PM_{10}s and the $PM_{2.5}$s have any independent or free-standing irritant effect. Professor Hay, on the other hand, believes that the role of these micro-particles is primarily, albeit not solely, as "porters" for irritant chemicals which become adsorbed onto them. Put in these terms, the divergence of view between the experts is minor. In any

event, in the circumstances of the present case I do not consider that this slight parting of the ways really matters, save perhaps in one respect. Whether or not the PM_{10}s and the $PM_{2.5}$s could in theory have any independent irritant effect, it must be obvious that virtually all of them operating in this smoke plume were vigorous and prolific carriers of no doubt numerous chemicals which were dispersed into the environs. I am prepared to accept that particularly the smaller fraction could have had a free-standing irritant effect, but it does not matter. The only point one should not lose sight of is that the quantities of acrolein, for example, have been separately modelled. This modelling includes all the acrolein, whether travelling on its own, or aided by a micro-particle. The possibility of double counting must be resisted.

148. Mr Kent advanced the more robust submission that I should be ignoring the micro-particles altogether. Given that they have little or no independent irritant effect, and given also that the acrolein and aldehydes have been separately modelled, there is no need for them as a surrogate; we have the real thing. Mr Kent relied on logic and on Dr Hind (his respiratory expert) in support of this submission.

149. I cannot accept Mr Kent's point. Scrutinising the micro-particles is merely a different way of trying to examine this vexed issue. It *"puts a handle"* on it, as both Mr Shillito and Professor Hay have agreed in their joint report. Mr Shillito also agreed that micro-particles could be used as a surrogate provided that due caution was accorded. For me, the issue is not relevance, but weight. The fact that there is direct modelling evidence of the aldehydes does not mean that it is wrong in principle to consider evidence of an indirect nature, particularly given that (a) the micro-particles will be porters for more than just the aldehydes, (b) the modelling evidence is not especially robust, and the more of it the better, and (c) there is a mass of academic literature which chooses to examine the micro-particles. Professor Hay suggested in addition that scrutinising the micro-particles betokens a conservative approach in any event because there must be numerous irritant chemicals travelling independently of them in the plume, and so modelling the micro-particles may underestimate rather that overestimate the global value of the irritants. However, in my judgment this is somewhat speculative, given that (a) many micro-particles may be free of irritant chemicals, and (b) no scientific paper has on my understanding sought to ascertain the likely ratio of micro-particles to chemicals of toxicological interest (*pace* the answer Dr Hind gave to my question). Without this data, one cannot really say how good or accurate a surrogate the micro-particles truly are.

150. Professor Hay's advice to me was to consider both the PM_{10}s and the $PM_{2.5}$s. Although CERC's recent plume modelling has not made much of the PM_{10}s, and the histograms have ignored them, I see no reason for excluding them from account. The Defendant is not remotely prejudiced because Mr Shillito has contributed fully to this discourse.

151. As with the irritant chemicals, the issue arises as to the appropriate threshold levels to take. The experts are agreed that the most authoritative domestic source is the report by the Committee on the Medical Effects of Air Pollutants ("COMEAP"), Review of the UK Air Quality Index. Although this report is concerned not with the effects of smoke but with ambient air quality in general, it provides considerable assistance with the issues I have to resolve. The authors observe that at low concentrations of particles the thresholds have an element of arbitrariness about them, and that it

remains uncertain whether any "safe" level exists. This is a matter which Mr Redfern explored with Professor Hay in evidence. However, the authors also state:

> "While we recognise the possibility that there is no threshold of the health effects of pollutants, nonetheless we consider that AQI can be developed that provides useful information on the possible effects on health at different pollutant levels in the short-term, and identifies individuals likely to be most susceptible.
>
> …
>
> The health response to increases in outdoor air pollution varies between individuals and sub-groups of the population … Individual susceptibility may affect the level at which health effects are noticed and the rate of increase in symptoms as air concentrations increase."

152. In any event, (a) the burden of proof is on the Claimants, not on the Defendant, and the best that Mr Redfern can do is persuade me to alight on appropriate threshold levels in view of all the expert and other evidence in this case; and (b) I cannot agree that *any* exposure is intrinsically unsafe. In answer to my question, Professor Hay accepted that *"you could come up with a value … that the majority of people would not be affected at low levels"*. Overall, Mr Redfern's submission amounts (in loose adaptation of Chaucer) to an impermissible attempt to make a virtue out of uncertainty.

153. The COMEAP report sets out two different sorts of threshold level. More valuable for present purposes are the trigger thresholds based on two consecutive hourly mean concentrations, subject to the second hourly mean concentration being greater than the first. Although these are principally warning levels, Professor Hay's advice to me, which I accept, is that they represent the sort of levels at which irritant effects might begin to be experienced, adopting I have to say a highly precautionary approach. I do not, however, accept Professor Hay's point that only one hour's exposure might be sufficient. I do not consider that COMEAP may properly be rewritten in this way, and it is also noteworthy that Professor Hay's point was specifically based on the premise that *"the particulates are only one small fraction of the material that will be arriving at somebody's respiratory tract"*. On the basis that the aldehydes represent the majority of this material, they have of course been separately modelled. At Table 3-2 of the COMEAP report (page 17 on the internal numbering), various suggested thresholds are advanced for the micro-particles, related to three bands, "moderate", "high or above" and "very high or above". Professor Hay did not say which of these bands I should select for present purposes, although he did in relation to the 24-hour mean thresholds. Doing the best I can on the available evidence, I take the "high" value for both PM_{10}s and $PM_{2.5}$s, namely 107 μg/m³ and 74 μg/m³ (as it happens, CERC's recent modelling adopts these values). These thresholds do not appear to discriminate between non-vulnerable and vulnerable individuals, but the precautionary principle would suggest that they are designed for the vulnerable.

154. The second series of threshold levels are the 24-hour mean levels for both micro-particles of interest, again set out in various bands. The experts were not agreed as to

which bands were appropriate, but I unhesitatingly prefer Professor Hay's evidence to Mr Shillito's. Professor Hay advised me to select the following thresholds from page 33 of the COMEAP report:

- PM_{10}s: 51 μg/m³ for vulnerable persons, 76 μg/m³ for the non-vulnerable.
- $PM_{2.5}$s: 36 μg/m³ for vulnerable persons, 55 μg/m³ for the non-vulnerable.

As before, this betokens a liberal approach, namely one favourable to the Claimants.

155. The COMEAP review also contains the following illuminating expository passages:

> "**Short-term effects of air pollution on health**
>
> Air pollution has a range of effects on health. However, air pollution in the UK does not rise to levels at which people need to make major changes to their habits to avoid exposure; nobody need fear going outdoors.
>
> ***Adults and children with lung or heart conditions.*** It is well known that, when levels of air pollutants rise, adults suffering from heart conditions, and adults and children with lung conditions, are at increased risk of becoming ill and needing treatment. Only a minority of those who suffer from these conditions are likely to be affected and it is not possible to predict in advance who will be affected …
>
> ***Older people*** are more likely to suffer from heart and lung conditions than young people and so it makes good sense for them to be aware of current air pollution concerns.
>
> ***General Population*** At very high levels of air pollution, some people may experience a sore or dry throat, sore eyes or, in some cases, a tickly cough – even healthy individuals … [page 90]
>
> **…**
>
> There has been very little work conducted on timescales of less than 24 hours and most knowledge of the acute effects upon health is based on day-to-day changes in air pollutant concentrations.
>
> The acute effects of particle exposure include increases in hospital admissions and premature death of the old and sick due to diseases of the respiratory and cardiovascular systems. The evidence is that both $PM_{2.5}$s and PM_{10}s cause additional hospital admissions and deaths on high pollution days. There are also less severe effects of short-term particle exposure during pollution episodes, such as worsening of asthma symptoms and even a general feeling of being unwell leading to a lower level of activity … [page 114]"

156. The final point which arises in the context of the COMEAP review is the salience of ambient background levels to the health threshold levels I have specified. This is a point which meshes with the plume modelling evidence, and may be expressed in these terms. If, for example, the ambient background level (i.e. the level untrammelled by the smoke plume) is X μg/m³ of PM_{10}s for any given period and location, and the modelled concentration of PM_{10}s is Y μg/m³ (ignoring the background level) for the same period and location, which value should be calibrated against the relevant health threshold level for these micro-particles? In my judgment, the answer is X + Y, applying the straightforward tortious principle of material contribution to the damage. There are average hourly background data from the Briery Hey monitoring station, some 1,250 m away from the Sonae plant. I assume for present purposes that the background levels do not vary much over the whole area under consideration, although they may be higher nearer the M57. Accordingly, these data will be used in conjunction with the 24-hourly mean concentrations of PM_{10}s (see paragraph 152 above) as evaluated for a limited number of Test Claimants (see paragraph 171 below).

157. Professor Hay drew my attention to an important paper authored by Kunzli et al, published in the American Journal of Critical Care Medicine in 2006, Health Effects of the 2003 California Wildfires on Children. The focus of this study was a vast, catastrophic series of forest fires in the State of California, visible from space. Local air quality monitors recorded hourly particulate matter concentrations approaching 1,000 μg/m³, being 10-20 times the typically observed ambient levels. The researchers collected data from questionnaires sent out by email within about one month and eight months of the fire (i.e. far sooner after the fire than the questionnaires in the present case, and unrelated to any medico-legal context). The children suffered a range of symptoms not at all dissimilar from those allegedly sustained in the instant case. What is of great interest, though, is the recorded 5-day mean PM_{10} exposures for children in the various locations identified in Table 2 of the paper. From this table it may be seen that for children with exposures in the range of 30-52 μg/m³, there were precious few complaints of symptoms. However, as will become apparent at a later stage in this judgment, the Californian cohort suffered far higher mean levels of exposure to PM_{10}s over a 5 day period than did their analogues, including adults, in the present fire. Symptoms were only experienced in significant numbers when the 5-day mean PM_{10} exposure level was in the 100+ μg/m³ range. To my mind, this paper significantly avails the Defendant and not the Claimants.

158. The final point which falls to be addressed on Professor Hay's evidence is the approach to be taken to what Mr Redfern described as "sub-threshold" levels of exposure. Perhaps anticipating that very few of his clients could demonstrate on the science that they suffered an "above-threshold" exposure, Mr Redfern was astute to draw out of Professor Hay evidence which might prove the Claimants' case on the balance of probabilities. Whereas it is quite true that many of the Claimants were exposed on the plume modelling evidence to the *odour* of acrolein, that is far from demonstrating that any of them suffered actionable personal injury. Indeed, if anything it is a point which tells against the Claimants owing to the impact of human suggestibility. If Claimant X smelt absolutely nothing, then s/he would probably have to be dishonest to bring a claim. No one is suggesting that the smoke plume contained odourless or occult properties akin to carbon monoxide. If the Claimants smelt

something, they might *believe* that they were exposed to a chemical which might have injured them in some way. I will be reverting to this point subsequently.

159. A "sub-threshold" exposure leads a Claimant nowhere unless either the science is wrong in some way, or the adverse health effects are additive or cumulative. I did not permit Professor Hay to perform the additions he obviously wished to, but I doubt whether my ruling truly impeded the Claimants (and, if it did, I am not reversing it). As I have pointed out, Professor Hay and Mr Shillito do not appear to be far apart in relation to the total aldehydes. I asked Mr Shillito whether he would be prepared to add a figure or percentage to his 500 μg/m³ threshold, in order to reflect the potential impact of other irritant chemicals in the mix. He suggested a figure of 5%. In the context of this case, that figure is *de minimis* and I ignore it. It falls within the general margin for error in the toxicological and plume modelling evidence. I have no other evidence before me on which to select any different figure. As for the possible cumulative effects, these are already addressed within the 24-hour mean thresholds. There is no proper basis on the available evidence for aggregating a series of below-threshold 24-hour means in order somehow to attain an above-threshold value.

160. Professor Hay was not asked to comment on a paper authored by Larrieu et al, Are the Short-Term Effects of Air Pollution Restricted to Cardiorespiratory Diseases?, published in the American Journal of Epidemiology in 2009. There, the authors examined over a six year period the possible effects of 10 μg/m³ increases in the ambient PM_{10} levels in relation to complaints of upper and lower respiratory tract diseases, headache and asthenia, skin rashes and conjunctivitis. Strictly speaking, therefore this study was not about the effects of smoke, but one might have thought that these would be *a fortiori*. The study found that there were relatively modest increases in complaints to GPs, ranging from 1.5% (for the respiratory tract) to 3.5% (for skin rashes, including dermatitis). However, it should be noted that the association for skin rashes was said to be "close to statistical significance" and not above it. Interestingly, Dr Larrieu's group pointed out that asthma was not associated with any of the indicators considered.

161. In my judgment, it would not be right to put much weight on this single study. The authors rightly observe that it is a unicentre study with a population which might have changed during the study period. I accept the evidence of Dr Iain Foulds that the results, even if valid, show only a slightly enhanced attendance rate at GP practices, that we have no evidence as to the severity, as opposed to the mere fact of the complaints, no indication of the extent to which those complaints were objectively validated, and no replication of these findings elsewhere.

162. In his closing submissions Mr Redfern, armed with this study and the separate WHO Guidelines, urged me to instruct further plume modelling based on a 10 μg/m³ increase in PM_{10} levels. I have declined this request. The WHO Guidelines, based on PM_{10} levels as low as 3-5 μg/m³, are concerned only with long-term health effects. In my judgment, evidence that increases as low as 10 μg/m³ might have significantly affected even some of the Claimants is extremely tenuous and speculative, and I prefer to proceed on the basis of the COMEAP data, setting out indicative health thresholds which are already favourable to them.

THE METEOROLOGICAL EVIDENCE

163. Fortunately, the meteorological evidence in this case is now agreed, and it is unnecessary for me to do more than quote from paragraph 18 of the Joint Statement of Mr Norman Lynagh and Dr Richard Wild:

> "On the basis of the 'best estimate' winds the following are dates and approximate times on which the wind would have been blowing from the Sonae factory towards at least part of the residential area or very close to it:
>
> 9th June: 22:45 – midnight
>
> 10th June: 00:00 – 08:30
>
> 12th June: 04:00 – midnight
>
> 13th June: 03:00 – 05:00
>
> 14th June: 06:45 – 09:15 and 14:30 – 20:30
>
> 15th June: 01:00 – 13:30
>
> 16th June: 02:00 – 05:15
>
> 17th June: most of the day
>
> 18th June: 00:00 – 01:15
>
> 20th June: 05:45 – 07:30
>
> 21st June: 00:00 – 01:45
>
> 22nd June: 00:00 – 06:00
>
> 24th June: 19:30 – midnight
>
> 25th June: 00:00 – 05:30
>
> 26th June: 01:00 – midnight
>
> 27th June: 00:00 – 11:00
>
> These are similar to the findings in our reports.
>
> …
>
> There were 6 days on which winds did not blow from the Sonae factory towards any residential area at any time. These were 11th, 19th, 23rd, 28th, 29th and 30th.

> There was only one day, the 12th, during which winds of any strength were blowing directly from the Sonae factory towards residential areas for an extended period of time."

164. The meteorological experts do not refer to July data, but the plume modellers have taken these into account. Plainly, the period of greatest interest is 10th – 13th June, when the emissions were at their highest levels. For stage 2 of the fire, the wind was blowing from the east, towards Kirkby, only for four hours (up to 08:30). For the first part of stage 3, the wind was blowing in that direction for 20 hours on 12th June and for 2 hours on 13th June. Given the wind direction, 17th June is also a date of some importance.

THE PLUME MODELLING

165. Nothing now separates the plume modelling experts in relation to the area of expertise with which they are strictly concerned. What previously divided them were the data or inputs apt to be fed into the same version of the ADMS computer model that each was using. Given that I have taken control of these variables, the computer programme must yield the same results whoever operates the programme.

166. I fully appreciate and understand that plume modelling is not an exact science. There is an element of inherent uncertainty inasmuch as the programme has its limitations; it cannot replicate the fabulous complexity of the real world. However, the degree of imprecision must not be over-stated, and I do not accept Mr Redfern's reference to "an educated approximation". Nor must it be assumed that the plume modelling underestimates indicative exposures. It is just as likely that it overestimates them.

167. I have already discussed aspects of the evidence of Dr David Carruthers in relation to emission factors and the overall methodology of plume modelling. In my view, he was a compelling and beguiling expert who had the knack of explaining quite complex concepts clearly, elegantly and with appropriate enthusiasm. I was not so appreciative of Mrs Angela Spanton, who gave the impression of lacking a degree of confidence and authority. I fully recognise and understand that it was always the Defendant's intention to call Dr Hall to speak to the reports of Envirobods, but unfortunately he died in February 2015. However, I do not accept the point that Mr Redfern made in cross-examination that Mrs Spanton must be incorrect in her assertion, based on extant modelling, that only 30% or thereabouts of all 16,000 plus Claimants were meaningfully exposed to the smoke plume at all. Looking at the location of the majority of the Claimants in relation to the A580 East Lancs road and the M57, it does appear that about 70% of the total cohort, some of whom were represented within the constituency of Test Claimants, were exposed only to minimal levels. Surpassing the acrolein odour recognition threshold for short periods comes nowhere close to being able to prove an adverse health impact. Dr Carruthers accepted in cross-examination that his modelling showed the same general picture.

168. Despite his compelling qualities as an expert, and above all his presentational abilities, Dr Carruthers was in my judgment significantly hampered, if not misled, by the information he was given by the Claimants' solicitors. Notwithstanding what is said in his first report, this was by no stretch of the imagination a fire which blazed for

16 days. Like Professor Hay, he was "drip-fed" information over the course of his litigation, much of it being inaccurate, unreliable or unsubstantiated. I should make clear that in no respect does the fault lie with Dr Carruthers. The internal assumption may have been that this case would settle, but there are obvious risks in not briefing experts properly.

169. As regards the Claimants' plume modelling evidence, the foregoing point may be illustrated in this way. CERC's first attempt at modelling was on the basis of nine separate factual scenarios, most of which were unsustainable (I accept that it was at least arguable that there were more than 1,550 Te of woodchips in the bunkers). That attempt was also predicated on a fire history/timeline which was unsupported by any expert evidence, and unsustainable in the face of the photographs. Once the Defendant's expert evidence was disclosed, what the Claimants needed to do was to regroup and instruct CERC to remodel indicative exposures on the basis of Dr Mitcheson's report and/or a total mass of 3,000 Te in the six bunkers (if the latter course were adopted, permission would have been needed to rely out of time on the evidence of a fire engineer to combat Dr Mitcheson). Moreover, the remodelling should have been done both generically and in relation to each Test Claimant. Although CERC carried out this exercise on an informal basis (based as it happens on Dr Mitcheson's data), and various data and histograms were disclosed during the course of without prejudice meetings with the Defendant's experts, these were not placed before the court on any proper basis until after the trial started. Fortunately for the Claimants, Mr Kent had the good grace and judgment not to object. That said, even if he had done I would have allowed this evidence in, notwithstanding its tardiness.

170. What is now available from the Claimants' side is the following:

- tables (showing the original 40 Test Claimants) examining the emission rates for $PM_{2.5}$s and PM_{10}s using the residential fireplace model for emission factors and the COMEAP trigger values of 74 and 107 μg/m³. Unhelpfully, the tables contain two sets of meteorological data, but helpfully they depict the number of hours of exceedances.

- tables (as before) examining the "total aldehydes" on the basis of the two emission rates (described by him as factors) originally proposed by Mr Shillito (although he is now content to proceed on the basis of the higher rates of 600 mg/s/MW) and a health threshold of 500 μg/m³.

- a similar table for acrolein based on Mr Shillito's emission rate of 114 mg/s/MW and the odour recognition threshold of 0.38 μg/m³.

- three separate tables focusing on the three highest exposed Claimants: these examine predicted hourly concentrations of PM_{10}s on alternative emission factors, and include the Briery Hey background values.

- standardised histograms for each of the 20 Test Claimants comparing their exposures hour by hour against five separate thresholds (the PM_{10} threshold has not been included).

- various contour plots examining different chemical/particles and emission factors.

171. The landscape has of course changed in the light of my findings. Shortly after the close of the trial, I invited CERC to undertake further modelling of the smoke plume limited to the 20 Test Claimants now under scrutiny, using agreed data where appropriate and the specific bases set out below, presented and/or tabulated as follows:

- a contour map showing the number of hours exceeding the acrolein odour recognition threshold of 0.38 μg/m³.

- a contour map showing the number of hours exceeding the lowest recorded health threshold for acrolein of 146 μg/m³.

- a contour map showing the number of hours exceeding the health threshold for "total aldehydes" of 500 μg/m³.

- a contour map showing the number of hours exceeding the PM_{10} trigger value of 107 μg/m³ (based on an emission factor of 27.5 g/kg for stage 2 and an emission factor of 12.5 g/kg for stage 3). I appreciate that when interpreting this contour map I will need to take into account the point that COMEAP requires two consecutive hours.

- a similar contour map for the $PM_{2.5}$ particles based on a trigger value of 74 μg/m³, and emission factors of 22.6 g/kg for stage 2 and 10.25 g/kg for stage 3.

- a table (showing only the 20 Test Claimants and the agreed meteorological data) examining exposures to PM_{10}s using my emission factors for stages 2 and 3 (viz. 27.5 g/kg and 12.5 g/kg) and the COMEAP trigger value of 107 μg/m³. As before, the table will show the numbers of hours of exceedances above this trigger value.

- a table (showing only the 20 Test Claimants and the agreed meteorological data) examining exposures to $PM_{2.5}$s using my emission factors for stages 2 and 3 (viz. 22.6 g/kg and 10.25 g/kg) and the COMEAP trigger value of 74 μg/m³. As before, the table will show the numbers of hours of exceedances above this trigger value.

- a table examining the "total aldehydes" on the basis of an emission rate of 600 mg/s/MW and a health threshold of 500 μg/m³. As before, the table will show the numbers of hours of exceedances above this trigger value.

- a similar table for acrolein based on Mr Shillito's emission rate of 114 mg/s/MW and a lowest recorded health threshold for acrolein of 146 μg/m³.

- a similar table for acrolein based on Mr Shillito's emission factor of 114 mg/s/MW and the odour recognition threshold of 0.38 μg/m³.

- separate tables for Test Claimants numbered 24 (10th and 12th June), 30 (10th, 11th and 27th June), 33 (work) (11th, 15th and 20th June), 37 (10th, 12th and 17th June) and 39 (10th, 12th and 17th June), using my emission factors and rates, and the agreed meteorological data, setting out the hourly concentrations of PM_{10}s and the Briery Hey background data for those dates.

- one set of standardised histograms for each of the 20 Test Claimants comparing their exposures hour by hour against the following separate threshold values: the odour recognition value for acrolein; the lowest recorded health value for acrolein, and total aldehydes (all based on the previously stated emission factors and threshold values).

- one set of standardised histograms for each of the 20 Test Claimants comparing their exposures hour by hour to the $PM_{2.5}$s and PM_{10}s, using the previously stated emission factors and rates, against the trigger threshold values of 74 and 107 μg/m³ respectively.

172. For the avoidance of doubt, I was not inviting CERC to deduct 20% in relation to the $PM_{2.5}$s and PM_{10}s to reflect moisture content. I was not asking CERC to model the hydraulic oil, the foam and the conveyor belts (the emission factors for these are not available, and they are being accounted for in a different way). As regards the 0.8% contamination, CERC was required to model this in the same way as it had done previously, in other words (on my understanding) taking emission factors for household waste. Finally, CERC's remodelling was carried out on the basis of my earlier findings in relation to total initial tonnage in the bunkers, heat release rates etc.

173. I appreciate that I had not specifically requested tabulation of CERC's remodelling against the 24-hour mean values for the PM_{10}s (51 μg/m³ for vulnerable persons, 76 μg/m³ for the non-vulnerable) and the $PM_{2.5}$s (36 μg/m³ for vulnerable persons, 55 μg/m³ for the non-vulnerable). However, when I gave my instructions to CERC I was confident that I would be able to ascertain the position as regards these values from the considerable array of data which I had requested, in particular the five most heavily exposed Test Claimants. In the event, CERC has kindly provided the 24-hour mean values for this sub-group.

174. The fruits of CERC's remodelling have been appended to this judgment (see Appendix 2) and are analysed further below.

175. Before leaving this section, I have to observe that there are features of the plume modelling which are not wholly satisfactory. One specific matter which concerns me is the ability of the model to provide a "handle" on subtle changes in wind direction and of temperature, and the possible ability of smoke to linger and seep. The model cannot account for the peripatetic nature of human behaviour, and in many cases it is difficult to know the exact position of any Test Claimant during daylight hours. On the other hand, the plume model fixes on external exposures, and people remaining indoors will have been exposed to less. Further, it is unlikely that most individuals' natural daily movements would have brought them closer to the Sonae plant; common sense would suggest that they would have moved in the opposite direction. Mrs Spanton referred to the "margin for error" within the model, but its nature and extent was not explored. Many of the sociological points set out above were made during the

course of the trial, but the inherent lack of precision of the plume model was not quantified. These concerns aside, the plume modelling is the best evidence the present state of human ingenuity can presently provide.

176. In my view, this is the now appropriate stage for me to set out the legal principles which govern these claims.

GOVERNING LAW

177. Most of the legal principles governing a claim of this nature are so axiomatic that explicit recognition in this judgment is not required. However, it is appropriate to address four specific areas.

178. First, it is common ground that the Claimants cannot recover damages for personal injuries unless they establish on the balance of probabilities that they sustained what the law regards as "actionable injury". It is insufficient for them to prove, without more, inconvenience and distress. I examined the relevant jurisprudence on this issue in my recent decision in Greenway and others v Johnson Matthey Plc [2014] EWHC 3957 (QB). Although, at the time of writing, I am aware that my decision is en route to the Court of Appeal, I am confident that my coverage of the general principles (as opposed to their application to the cases then under consideration) is uncontroversial. The leading authorities are Cartledge v Jopling [1963] AC 758 (HL) and Rothwell v Chemical and Insulating Co Limited (2008) 1 AC 281 (HL). At paragraph 26 of my judgment in Greenway, I said this:

> "On my understanding of its reasoning, the House of Lords in Rothwell did not seek to reinterpret its earlier decision in Cartledge, although – as has been pointed out in the parties' written submissions – their Lordships' individual formulations of the legal test varied slightly. For Lord Hoffmann the test (in tort claims generally) involved the *"abstract concept of being worse off, physically or economically, so that compensation is an appropriate remedy"* (at 289D). For Lord Hope, the test was whether there was *"real damage, as distinct from damage which is purely minimal"* (at 297E). For Lord Rodger, the test was *"material damage"* (at 311F). It might be argued that some of the formulations tend to circularity. What may be more valuable is to consider how the test was applied to the facts of both Cartledge and Rothwell itself."

179. The facts of both Cartledge and Rothwell are far removed from the present scenario, and in the passage I have set out above I have alluded to the potential circularity which arises. Ultimately, in my judgment, this must be a question of fact and degree. A transient, trifling, self-limiting, reversible reaction to an irritant is not "actionable injury" for the purposes of the law of tort. These could fairly be described as "normal physiological responses". However, the ubiquity of this concept cannot be recognised because it could be used to characterise the reaction of human tissue to hydrochloric acid. The normality of the burn does not rob the injury from its characteristic of being actionable. In my judgment, if the degree of irritation is severe enough, whether or not

it becomes a pathological response expressed in terms of inflammation, it may be possible to hold that the line has been crossed. This is more likely to be so if there are several severe irritant responses occurring simultaneously: i.e. to the eyes, the nose, the respiratory tract, *and* they last long enough. Usually, one would expect to see evidence of a resulting inflammation in such cases.

180. The question does arise of whether a series of minor, sub-threshold irritations is capable of overleaping the bar set by the concept of actionable injury. Could 1,000 sneezes amount to a claim whereas 1, or even 10, sneezes plainly could not? I accept that difficulties might arise at the margins (imagine 1,000 consecutive sneezes), but ultimately this point collapses into what is in common parlance called the "zero sum game" rather more elegantly expressed by Lord Hope in Rothwell (see paragraph 42 of his opinion).

181. Having regard to all the available evidence, I intend to adopt a broad, common-sense approach to this issue. The clinicians differentiate between irritation and inflammation ("... -itis"), but my approach will not be strictly tethered to the strict medical or pathological concepts, although will recognise that these have some general utility. However, there is still a hurdle which each Test Claimant has to surmount.

182. Secondly, the Claimants seek to argue that it is sufficient for their purposes that they are able to prove that the Defendant's breach of duty materially contributed to the risk of injury. I was taken to the pre- and post-Fairchild jurisprudence, as well as to a number of decisions of the highest authority dealing with industrial disease. On this issue I am able to be quite brief, because in my judgment Mr Redfern is seeking to lead me into frank error.

183. In the present case we have one tortfeasor and, putting to one side for the time being questions of pre-existing vulnerability and background air pollution, one package of potentially causative agents. The issue is whether that package of chemicals and particles caused or materially contributed to the Claimants' alleged personal injuries. The Claimants do not have to prove sole cause, but they do have to prove material cause. It is conceptually and legally incoherent in a case such as the present to speak in terms of the smoke plume making it more probable that the Claimants might have suffered personal injuries, or (to put the same point in another way) that their risk of suffering personal injuries was increased. The issue is a binary one: either, on the balance of probabilities, they sustained an injury in consequence of tortious exposure, or they did not.

184. The Fairchild doctrine of "material contribution to the risk" is designed to cover two situations. The first is where there are two or more tortfeasors, and medical science cannot say which caused the injury. In order to achieve justice for the Claimants, the law relaxes the "but for" test of causation. However, in that situation it is beyond dispute that (a) the Claimant suffered personal injury, and (b) that injury was caused by a tortious agent (whose agent being the issue). The second situation is where there is one tortfeasor and two potential causative agents (one "guilty" and the other "innocent"), but it cannot be proven which actually caused the Claimant's injury. Again, in all these situations it is beyond dispute that (a) the Claimant suffered personal injury, and (b) the industrial agent was capable of causing that injury.

185. I reviewed most of the relevant jurisprudence in Heneghan v Manchester Dry Docks Ltd and others [2014] 4190 (QB), another case en route to the Court of Appeal, but where my same general observation applies (see paragraph 178 above). The *locus classicus* remains McGhee v NCB [1973] 1 WLR 1. In "innocent" and "guilty" dust cases, the law does not require strict probabilistic proof applying the "but for" test. Mr Redfern drew my attention to the earlier decision of the House of Lords in Gardiner v Motherwell Machinery and Scrap Co Ltd [1961] 1 WLR 1424, another case where the first opinion was given by Lord Reid. Mr Gardiner claimed damages at common law for exposing him to conditions liable to cause dermatitis, and in failing to provide proper washing facilities. Lord Reid explained the basis for the plaintiff's recovery of damages for breach of duty in this way:

> "I can now sum up my view in this way. The appellant never suffered dermatitis before he was exposed during his employment by the respondents to conditions liable to cause that disease. His original symptom – an outbreak on the back of his hand – is admittedly typical of industrial dermatitis …
>
> In my opinion, when a man who has not previously suffered from a disease contracts that disease after being subjected to conditions likely to cause it, and when he shows that it starts in a way typical of disease caused in such conditions, he establishes a prima facie presumption that his disease was caused by those conditions. That presumption could be displaced …"

Other members of the Appellate Committee analysed the case on the basis that the appellant had proved his case on all the available medical evidence. Thus, Lord Reid's approach, albeit immensely authoritative, should be regarded as evidencing a minority view. This is the same minority view that Lord Wilberforce espoused in McGhee (see 7E), and in my judgment should be regarded as apart from the mainstream.

186. The fundamental reason why the "material contribution to the risk" principle, in any of its manifestations, cannot avail the Claimants is that it is incumbent on them to prove on the balance of probabilities that they were within the relevant envelope of material risk *as that concept is properly understood*. It is insufficient for them say – whatever the strength of their personal conviction may be – that they were at risk in the sense that they had *some* exposure. On that argument they should succeed even if the exposure were minuscule, measurable only in parts per trillion. In my judgment, any Claimant was only at risk if s/he can prove exposure at a level which was capable of causing personal injury. This the relevant risk for these purposes. For many people, exposure at these levels would not in fact cause personal injury, but the threshold defines the level at which they are at least "at risk". The sub-threshold terrain may not be "safe", in the sense that it cannot scientifically be proven to be 100% safe, but as I pointed out to Mr Redfern when he was examining Professor Hay on this topic, the *null hypothesis* works the same way in science and in law; all that varies is the standard of proof.

187. I should not be understood as being necessarily wedded to threshold levels which are derived from experimentation and scientific inquiry. The levels I have selected as

indicative are, in fact, favourable to the Claimants, but there remains room for an argument that they should be relaxed yet further to reflect the scientific uncertainties which abound, and the difference between the level of proof the law as opposed to science requires. The extent to which I will succumb to that argument depends on balancing the strength of the scientific evidence against that of the lay evidence. In the final analysis, however, I will have to arrive at appropriate thresholds, even if they are lower than those a purely scientific approach would mandate. To do otherwise would not be to exercise the "complete autonomy" which Mr Redfern suggests I possess, but to indulge in unprincipled decision-making. Whatever the final position on the thresholds, the position remains the same: below them no Claimant is within the envelope of risk.

188. The third legal issue which arises avails the Claimants, and there is no dispute about it. Given that a Claimant does not have to prove more than that the smoke plume materially contributed to his or her personal injury, success is achieved either if it is shown that exposure exacerbated a pre-existing condition (even if a person without that condition would not have sustained any injury), or if exposure, added to background levels of pollutant, took that Claimant above a relevant threshold (even if the background level was sub-threshold).

189. The fourth legal issue which arises is the difference between the legal and scientific standard of proof. The Court of Appeal has stated on a number of occasions that there is a difference between these two standards (see, for example, the judgment of Smith LJ in MoD v Wood [2011] EWCA Civ 792), but what is more difficult is to specify exactly what it is. Without attempting an academic or jurisprudential analysis in a case already bristling with difficult issues, it seems to me that the relevant points may be made in this way. First, a scientist would tend to discount retrospective accounts given some considerable time after the events in question. A lawyer might treat such accounts with condign appreciation, depending on their overall reliability, credibility and consistency, because the approach of the common law has always been more inclusively flexible than a purely scientific approach. So, the fact that the lay evidence in the present case would not get past the front door of any reputable scientific journal is not quite the point. Secondly, the evidence supporting the various health threshold levels in the toxicological literature may have been filtered through more robust and stringent filters than those which would be required by a lawyer applying probabilistic standards (on the other hand, I repeat the point that many of the COMEAP levels are precautionary). Thirdly, there are respects in which the science is inherently uncertain, notwithstanding its claim to precision. I have in mind the plume modelling evidence. A scientist would say that if the modelling fails to prove the case, that must be the end of the matter. A common lawyer would say that there remains room for flexibility and reasonable latitude, and that the whole picture must be held in mind. How much room, though, needs very carefully to be considered.

190. Mr Redfern drew attention to the decision of the Court of Appeal in Armstrong v First York Ltd [2005] 1 WLR 2751. In that case, the claimants alleged that they suffered soft-tissue injuries to their spines in consequence of a relatively low-impact road traffic accident. The parties relied on accident reconstruction evidence based on second-hand information about the damage the vehicles had sustained. This evidence directly contradicted the claimants' evidence and stated that they could not have sustained injuries as they alleged. The trial judge found the expert evidence to be

convincing and the claimants to be blameless and honest witnesses. The judge preferred their account. The issue which arose on appeal was whether the trial judge could have rejected the expert evidence without finding any flaw in it. The Court of Appeal held that he could: he had weighed up all the evidence in the case, and was entitled to conclude that the claimants were not lying and that there had to be some inaccuracy in the expert's evidence.

191. At paragraphs 26 and 29 of his judgment, Brooke LJ said this:

> "In my judgment, in this very difficult case the judge directed himself correctly as a matter of law. He was entitled to consider the evidence he had been given by the Claimant extremely carefully, directing himself about the dangers of witnesses who may seem to be plausible but in fact are telling a pack of lies, and directing himself to consider very carefully the evidence given on behalf of the defendant. He formed the view that he could not be satisfied that these witnesses were telling a pack of lies. He was very impressed by their evidence, and he concluded, when he had to balance the evidence of each side, that there must be - although he accepted fully that he could not say what it was – something that was not accurate in Mr Child's evidence in that particular case.
>
> …
>
> In my judgment, if we dismiss this appeal in this case we are not opening the door to a whole lot of dishonest claimants to recover just because there may be cases in which the honesty and force of a claimant's evidence impresses a trial judge in the way the evidence of these claimants did on this particular occasion. In very many cases the evidence of a witness like Mr Childs may well be sufficient to tip the balance strongly in the defendant's favour."

192. Brooke LJ also referred to the decision of the Court of Appeal in Coopers Payen Ltd v Southampton Container Terminal Ltd [2004] 1 Ll Rep 331, where Lightman J said this:

> "There is no rule of law or practice in such a situation requiring the judge to favour or accept the evidence of the expert or the evidence of a witness of fact. The judge must consider whether he can reconcile the evidence of the expert witness with that of the witness of fact. If he cannot do so, he must consider whether there may be an explanation for the conflict of evidence or for a possible error by either witness, and in the light of all the circumstances make a considered choice which evidence to accept. The circumstances may be such as to require the judge to reach only one conclusion."

193. The context of Armstrong differs from the present case in a number of respects. There, the choice was stark and binary for the trial judge: the claimants were either

truthful or they were liars. No witness may be heard to contradict Newton's Laws; but the science – in Armstrong, the engineering evidence – unlike Newton, might have been flawed.

194. In the present case the situation is far more complex. There is a mass of science, of variable inherent weight across the board, and a mass of lay evidence. Earlier sections of my judgment have addressed the science, but I am about to move onto the evidence of the clinicians and the Test Claimants. My approach will be to assess that evidence on a traditional, common law basis, and finally in the concluding section of my judgment I will attempt the syncretism that Lightman J recommends. If that reconciliation cannot be achieved, each corpus of evidence will continue to be judged in its own right, but at the end of the day something may have to yield.

THE EVIDENCE FROM THE CLINICIANS

195. Although this body of evidence was called after I heard from the Test Claimants, I consider that it is appropriate to examine the generic aspects of the evidence from the clinicians at this stage.

196. I heard evidence in four disciplines: lower respiratory tract; upper respiratory tract; skin; and eyes. All the medical experts underlined the importance of taking a detailed and accurate history from a Claimant.

197. Dr Christopher Hardy FRCP, consultant general and respiratory physician working primarily at the Manchester Royal Infirmiary, was called by the Claimants. Dr Charles Hind FRCP, consultant physician working primarily at the Liverpool Heart and Chest Hospital, was called by the Defendant. Temperamentally very different though they clearly were, their expertise and experience is immense and I was extremely grateful for their evidence.

198. The respiratory physicians examined the then 40 Test Claimants jointly in November 2014 and provided separate reports for the Court. They also provided a Joint Statement following a without prejudice meeting in the usual way. Although the dates of examination varied slightly, the pattern was the same in relation to the other disciplines.

199. In a powerful and sustained piece of oral evidence, Dr Hardy explained why he considered that the Test Claimants he saw were likely to have been victims of exposure to the smoke plume, causing symptoms to the lower respiratory tract. In his view, the cases tended to follow a clear pattern. Many of the people were aware of the fire, owing to its terrible smell and taste, which were new. Some of them described a very large black cloud crossing their homes and gardens. At least one person showed the doctors a photograph on his phone of the cloud in relation to his garden. All decided to batten-down their houses, but the smell and taste persisted. Generally, within a day or two, the Test Claimants developed symptoms. The first thing they tended to describe was itchy eyes, of varying degrees of severity. Nasal symptoms were described by virtually everyone: their noses were either very blocked or very runny. Sore throats were very prominent. Virtually all of them would say that their throats were very dry at night, and that they needed a glass of water by their beds to

keep their throats moist. Claimants also complained of a severe cough with tenacious sputum, sometimes with breathlessness and wheeziness, and some had skin symptoms.

200. According to Dr Hardy, the differences in terms of the histories taken related principally to the duration of symptoms. In relation to those who had pre-existing problems or issues (e.g. asthma; COPD; smokers), the onset of symptoms was different as well as their offset. It was difficult to time this precisely, but as regards those who had no co-morbidity the symptoms tended to last 3-6 weeks; in relation to those who did have other problems, the duration of symptoms could be measured in months. Accordingly, in Dr Hardy's view, these were severe symptoms, albeit temporary.

201. Dr Hardy addressed the possibility that some, many or all of the Test Claimants might have suffered from a coincidental viral infection. He agreed that there was a significant overlap between the presenting signs and symptoms of viral illnesses, and the Claimants' complaints of smoke exposure. Dr Hardy drew to my attention the inherent implausibility of so many people suffering from a viral illness (one might add, in the early summer), and he also referred to the very similar time period of onset and what he called the constellation of symptoms.

202. Dr Hardy explained that when exposure finishes, it will take time for the patient to recover. Smoke irritated and inflamed the mucosa, the lining of the respiratory tract, and ultimately the airways with the development of bronchitis. When the exposure ceases, the glands producing sputum will not turn off like a tap, as he put it.

203. Dr Hardy therefore felt that on the balance of probabilities, if a Claimant had been exposed to the smoke plume, and developed exactly the same symptoms as the others, then those symptoms were due to the Sonae fire.

204. Dr Hardy's understanding of the fire was that it was burning well for a week, and then smouldering thereafter.

205. Under cross-examination, Dr Hardy agreed that no definitive test existed to prove that these symptoms, if they were truly experienced, were due to the fire. He said that irritant chemicals can create an inflammation if the dose is sufficiently severe. He did not agree that people would fall ill at once; it might take up to 24-36 hours. In relation to the toxicological thresholds, he made the point that people may respond very differently to the same dose.

206. Later in his evidence, Dr Hardy said that people with underlying lung disease who suffer an inhalation injury would be more likely to develop an infection, because their immunological response will have been compromised.

207. Dr Charles Hind said that there was considerable common ground between him and Dr Hardy. The reaction of the lungs to an insult will depend on its severity. The irritant effects occur within seconds, or at the most minutes, of exposure. The noci-ceptors in the upper respiratory tract detect the presence of irritants and operate like "smoke detectors". The mucosal linings will become moist, and in due course the goblet cells will secrete mucous, producing mild physiological effects such as coughing, phlegm and tightness of the chest. Dr Hind stated that more severe and

prolonged exposure may result in acute inflammation of the lining of the respiratory tract, which could cause symptoms such as a persistent cough, difficulties in breathing and bronchospasm. Even in these cases the recovery period is likely to be short.

208. In Dr Hind's view, the difference between the irritant and the inflammatory response is likely to be a question of degree. He said that the health thresholds suggested by the toxicological evidence (e.g. 146 μg/m³ for acrolein) might be the level at which some people would begin to experience an irritant response. Dr Hind could not say at what level an individual might begin to be at risk of an inflammatory response.

209. Dr Hind also suggested that in June/early July the pollen count would have been moderate to very high (the nearest monitoring station is at Rotherham). He agreed that asthma sufferers could well respond at lower exposure levels.

210. In cross-examination, Dr Hind agreed that there were many vulnerable groups who might require a lower dose before experiencing symptoms. In general terms, he agreed that there was considerable individual variation, and that such variation was also seen between asthmatics (Dr Hardy had told me that asthmatics can vary in reactions to particular irritants by a factor of 40). He did not agree that smokers formed such a group. He said that if the evidence did not show significant or above threshold exposures to the smoke plume, one would need to examine alternative explanations for these complaints.

211. Dr Hind agreed with Dr Hardy that smoke exposure had the potential to lead to an infection because the microphages might be overwhelmed. However, I interpreted his evidence as suggesting that this might arise only if the body has already shown an inflammatory response to the irritant chemical. This chimed with Dr Hardy's evidence that there would need to be *"a lot"* of inflammation. It is very difficult to see how milder responses might induce infections.

212. I have set out the evidence of Dr Hardy and Dr Hind in some detail because (a) there is little between them, and (b) the generic points they make were mirrored in the evidence given by other clinicians. To my mind, four points of general application need to be emphasised. First, the difference between irritation and inflammation is likely to be one of degree, particularly at the margins. Secondly, human variability occupies a significant range. Thirdly, the strength of Dr Hardy's mini-foray into epidemiology must depend at the very least on the quality of the evidence, including the histories, elicited from all the Test Claimants. Fourthly, and perhaps most importantly, much of these experts' clinical opinion depended on their chosen point of departure. If the starting-point is exposure to smoke at levels which may have been sufficient to yield symptoms, then Dr Hardy's patterns and constellations become a compelling explanation for the phenomenon under scrutiny; and, moreover, evidential weaknesses in an individual Test Claimant's case became less important, because they may benefit from the similarities within the herd. If, on the other hand, the starting-point is exposure to smoke at levels insufficient to yield symptoms, then certainly the medical scientist would say that the quest for alternative explanations is necessitated. At the very least, a close examination of the individual circumstances of the Test Claimants becomes more important.

213. Moving on now to the upper respiratory tract, the forensic contest here was between Mr Andrew Swift FRCS, consultant ENT surgeon and rhinologist, primarily working

at the Aintree University Hospital (for the Claimants) and Mr Andrew Parker FRCS, consultant ENT surgeon, primarily based at the Royal Hallamshire Hospital, Sheffield (for the Defendant). As with their lower respiratory tract colleagues, I was greatly appreciative of their evidence.

214. Mr Swift had seen the DVD of the fire, and told me that he would have expected consequences from it, namely some degree of inflammation in the upper respiratory tract. The main insult would be to the nose, but the throat and larynx might also be entailed. Mr Swift's explanation of the nature of the irritant, and then the inflammatory responses, was very similar to his colleagues' in the related field, and need not be repeated.

215. My interpretation of his evidence was that Mr Swift did not accept that those who had suffered irritant responses would be more likely to experience head colds. He believed that this would not be so, otherwise hay fever suffers would succumb more readily to these. I did not understand Mr Swift to accept that the position was necessarily the same as regards those who suffered extensive inflammatory responses.

216. Mr Swift saw 14 Test Claimants, and believed that there was a commonality of complaints. The likely explanation for them would be irritation from an external source. In the majority of those he saw, he diagnosed rhinitis and pharyngitis.

217. Under cross-examination, Mr Swift agreed that his diagnosis of inflammation was based solely on the history he was given as to clinical symptoms, and on his clinical opinion, not on biopsy. He also agreed that any inflammatory response would be presaged by sensory irritation, in particular by sneezing. Mr Swift said that if an individual has an inflammatory response, and then is removed from the external source, it would take days, or more likely a couple of weeks, for there to be full recovery.

218. Mr Swift was a diligent, careful witness who was rightly concerned not to over-state the Claimants' case. Mr Andrew Parker was an engaging witness with a flair for succinct, apposite explanations. Thus, in relation to one of the *leitmotifs* in this case, being the difference between the irritant and the inflammatory responses, he said this:

> "Well, it depends upon how much irritant there is. If there is a significant irritant, then there will be an [immediate] irritant response. If there is more irritant, then it will turn into an inflammatory response, which is a pathological step change. Essentially, your Lordship, an inflammatory response is a final common pathway that the body has in relation to potentially injurious incidents, one of which is an irritant up the nose."

219. In Mr Parker's view, the nose is designed to filter out material which could be injurious further down. An irritant response will settle down very quickly after the irritant has gone, or has been deactivated by the nose. Particulates will be washed away by mucous, or can be swallowed. Later in his evidence in chief, Mr Parker said this:

> "Q. So given the understanding, perhaps with the benefit of Mr Redfern's description of the fire, of what the claimants you

examined experienced, would you have expected any of them, once the irritant was removed, to have spent as much as a week or two weeks or three weeks symptomatically?

A. I would say on the balance of probability, not. If the alleged exposure was for a period of a few days -- a period of a few weeks, then it's conceivable that there has been inflammatory change in the nose during that time because the exposure has been continuing.

Q. So are you contemplating a consistent level of concentration of the irritant?

A. The injurious agent would have to be present in the air in sufficient and significant dose for me to endorse that concept."

220. Under cross-examination, Mr Parker conceded that Claimants might have sustained an inflammatory reaction if sufficient exposure to the smoke went on long enough. In that sense, he was conceding very little: this is a truism. He did not accept that constant sub-threshold exposures could have had an additive component, but he fairly said that *"there might be a range of opinion on that"*. I will bear this in mind in due course, but in terms of the science of toxicology I have already said that I consider that possible additive and cumulative effects are addressed within COMEAP, and that unless the science is ignored, qualified or rewritten for the purposes of this judgment, one really should not go any further. Mr Parker's final view on this aspect emerged after questioning by me:

"MR JUSTICE JAY: But I think what is being put is each time you go in there will be a sub-threshold exposure, but it will happen often enough and in sufficient temporal proximity. In other words it's ongoing, but ebbing and flowing always below sub-threshold. There will come a point at which, bang, you start to get symptoms, not because you're sensitised, but because there's a sort of build-up. I just want to know what your reaction is to that.

A. My reaction is, on the balance of probability, I wouldn't expect that. There would have to be sufficient exposure to maintain a continued inflammatory reaction."

221. Mr Parker accepted in cross-examination that the absence of permanent damage does not preclude the possibility of a prior inflammatory response. He also accepted that it might take time for symptoms to resolve as the concentration of the alleged irritant falls. Finally:

"If the claimant has not been exposed as alleged then any symptoms that they report cannot be arising as a result of the alleged index incident. On this basis they will be unrelated to it and arising as a result of other mechanisms."

222. The dermatological expert evidence was given by Dr Paul August FRCP, consultant dermatologist, primarily working at the Leighton Hospital, Crewe (for the Claimants) and Dr Iain Foulds FRCP, honorary consultant dermatologist, based at the University of Birmingham (for the Defendant). Extremely helpfully, these experts were able to encapsulate areas of consensus and disagreement in a Joint Statement dated 3rd June 2015.

223. These experts made the same general points as did colleagues elsewhere. They were in agreement that there is no literature bearing on the question of possible contact dermatitis through smoke irritation. I have examined the Larrieu paper at paragraph 160 above. Dr August pointed out that the difficulty and cost of obtaining such evidence heavily militate against it, although he was compelled to accept that skin symptoms were not mentioned in the Californian Wildfires paper.

224. Dr August told me that the present focus is on a number of similar conditions, varying to some extent in nature and degree, namely puritus (itching), purigo (lesions), urticaria (an irritable condition of the skin caused by release of histamine) and dermatitis (an inflammatory condition which can be caused by contact with an irritant). A number of the Test Claimants also complained of acne excoriae and rosacea, which on my understanding are predominantly constitutional conditions which could be aggravated by exposure to irritants.

225. Despite the absence of evidence in the literature, Dr August said that he believed that about 1,000 out of the total cohort of 16,000+ Claimants were complaining of skin symptoms. That corresponds with the known proportion of the population which is vulnerable. However, in my view that piece of evidence taken in isolation cannot avail the Claimants in any way – the issue surely is, vulnerable to what? In any event, we know next to nothing about the non-Test Claimants.

226. Under cross-examination, Dr August accepted that timing of onset of symptoms, and levels of the modelled dose, were extremely important considerations. He, as did others giving evidence for the Claimants, accepted the possibility of cumulative exposures. He did not accept that onset would have to be immediate, but he did envisage that the probable time period for onset was likely to be in the region of hours to days. He agreed that the eyes were more sensitive than the skin, and he said that if the airways were safe, he would have thought that the skin would also be safe. Dr August accepted, as he was bound to, that the conditions of this litigation were removed from those of a scientific study, owing to the confounding factor of individuals claiming compensation serving to magnify recall bias.

227. Dr Foulds's evidence was of a piece with those in other disciplines testifying for the Defendant. In a powerful passage in his evidence in chief, he said this:

> "Well, we talked about irritant contact dermatitis, and irritants you can divide into absolute irritants and relative irritants. Absolute irritants will cause damage to the skin with one or two exposures. So, for example, hydrofluoric acid, if you pour that on your skin, is going to drill a hole in your skin. That's an absolute irritant, whereas relative irritants work by damaging the lipid cell surface membranes within the skin, causing damage to the integrity of the barrier function of the skin

> cumulatively over a period of time. So irritants are anything which dries the skin, degreases the skin, chemically attacks the skin over a period of time. So in practice exposure to irritants usually requires months or years of repeated exposure to damage the barrier level of the skin to cause problems, to break through the threshold of the actual barrier function of the skin to actually cause dermatitis. Now, most dermatitis will occur from cumulative damage unless that individual has had repeated exposure over a period of time just beneath the threshold and then the final insult will take them through the threshold. But in practice you need months or years of repeated exposure to develop irritant dermatitis, and that irritant dermatitis has to occur on areas which are susceptible. So hands from a wear and tear point of view, or if you're talking about an airborne irritant, the first place to get affected will be the eyelids, around the eyes and on the face. Yet none of the people that we examined had problems on their eyelids. So from irritation, I think it pretty well rules that out as a possibility. If it's an airborne thing, it's got to affect the areas exposed to the skin. Airborne irritants, airborne allergens where people become allergic to chemicals, affect the skin that is exposed to the air and not on covered sites. Yet a lot of the individuals we examined had problems on covered sites as well."

228. In his closing arguments, Mr Kent relied heavily on Dr Foulds' evidence about what the latter called the *"me too"* syndrome, which is particularly prevalent in industrial or potential product liability cases. For example, close investigation of an alleged problem (e.g. contact dermatitis caused by a "new system" wash-powder) demonstrated that very few individuals, if any, had problems genuinely attributable to the agent under scrutiny. Their causes were shown to have been unrelated.

229. Under cross-examination, Dr Foulds was asked whether he derived comfort from the Envirobods and Shillito evidence appearing to show that the Claimants were not sufficiently exposed. He said not, and contended that the stronger point here was the absence of a common factor or a common clinical picture. Dr Foulds said that there was a contradiction between the symptoms as they described them and the histories that were elicited. Most said that their symptoms cleared up within a short space of time. Dr Foulds denied that those with pre-existing conditions were more vulnerable.

230. The only generic issue on the dermatological evidence which I need resolve at this stage is whether exposure to smoke is capable of causing contact dermatitis. Professor Hay has advised me that there is no evidence that it can, and I have already discussed the Larrieu et al paper. In my judgment, the evidence base in support of the proposition that exposure to smoke at relatively low levels (i.e, at the sort of levels suggested by the plume modelling evidence) can cause dermatological problems must be regarded as extremely sparse, based on little more than clinical impression alone.

231. Finally, I heard evidence from two distinguished consultant ophthalmologists, namely Mr Louis Clearkin FRCS, working primarily at the Arrowe Park Hospital in the Wirral (for the Claimants), and Mr Ian Marsh FRCS, primarily working at the

University Hospital, Aintree (for the Defendant). Mr Clearkin described himself as a *"jobbing ophthalmologist"*, perhaps with a modicum of self-deprecating irony. I appreciated his charm and sense of humour.

232. All the Test Claimants the ophthalmologists examined had symptoms of tear film instability due to underlying meibomian gland dysfunction. These glands reside in the lid of the eye and are involved in the production of tears and mucin. Tear film instability may result when the external environment of the eye becomes abnormal in some way, causing tears to evaporate or become otherwise dysfunctional and the tear film to break down. In Mr Clearkin's view, exposure to the smoke has brought forward symptoms of tear film instability by 3-5 years. He put the point rather compellingly in this way:

> "I work on the Wirral. It's a very different area. Very refreshing to meet the good folk of Kirkby. You know, solid people, gave a proper account of themselves, pleasure to meet some of them, pleasure to meet all of them to a degree. As I say, I've never been to Rodney Street before. It was a life changing experience in many ways, but certainly it was a good gig in terms of dealing with patients. This was a very straightforward experience.
>
> Q. I wasn't asking about your pleasure.
>
> A. I do apologise.
>
> Q. I was asking how you found the group as far as the presentation of their conditions.
>
> A. It made me wish I worked in Kirkby and had much more straightforward people to deal with than on the Wirral. But I hope nobody has written that down. But they're very straightforward people. They struck me as no side to them, gave an account that I found, with one exception, one minor exception, entirely straightforward. They told me a story that fitted in with their clinical signs. I hear this story half a dozen times at clinic in terms of symptomatic tear film instability.
>
> Q. Due to exposure to smoke?
>
> A. No, due to a variety -- it's a common final pathway disease. Many things can precipitate symptomatology.
>
> Q. Smoke is one of them?
>
> A. Smoke is one of them. Sunburn, for example. A particularly dry day. A particularly cold day. It doesn't take much. It's a complex mechanism. It's very, very robust. When it goes, it goes."

233. Under cross-examination, Mr Clearkin agreed that he was making an implied assumption about the levels of smoke exposure being sufficient. He said that an

irritant chemical, for example the sulphur compounds produced by an onion, might provoke tears, which might wash away the tear film components, which might then precipitate chronic symptomatology. Ultimately, however, Mr Clearkin's evidence was that the posited environmental trigger is the final insult in the causative chain, the metaphorical straw which breaks the camel's back.

234. Mr Ian Marsh did not disagree with the general principle of tear film instability; his dispute with Mr Clearkin was as regards its causes, and as to the reliability of the Test Claimants' accounts. Unlike Mr Clearkin, he deferred to me on this last aspect. Mr Marsh emphasised that we have no antecedent examination of these individuals before the alleged exposure; all we have is what may now be observed four years after the relevant events. Mr Marsh did not accept the possibility of cumulative effects: if those were a real phenomenon, one would have expected a predominance of patients coming to ophthalmic clinics with the signs and symptoms of this dysfunction, having sustained only small doses of an irritant. Mr Marsh did not disagree with the general principle of one final insult pushing a patient over the edge as regards meibomian gland dysfunction, but could not endorse the proposition that the acceleration might have been by as much as 3-5 years. Nor did he accept that it was other than speculation that smoke should have accelerated a chronic condition.

235. Under cross-examination, Mr Marsh told me that there were some similarities, and also some differences, as to the various Test Claimants' symptoms and their onset. He agreed that by and large they presented with the symptoms that one would expect from exposure to large quantities of smoke.

236. I found Mr Marsh to be a somewhat dour witness who appeared to have been discomfited by the forensic process and Mr Clearkin's no doubt well-intentioned jibes about his consulting rooms. I cannot accept his contention that because the Test Claimants' evidence is "*subjective*" it cannot carry any weight. Rather, it is a factor to be weighed in the overall evidential mix. Even so, I have no hesitation in preferring his evidence over Mr Clearkin's at least as regards the sole generic issue that I am choosing to resolve at this juncture. I cannot accept the assertion that exposure to smoke could have accelerated symptoms by anything like 3-5 years. The notion that smoke might advance symptoms is theoretically possible in extreme cases, but is not really supported by other than somewhat speculative, assertive evidence. These Claimants are all likely to have had pre-existing tear film instability which ebbed and flowed in the ordinary course of the condition. I accept the possibility that a few developed this dysfunction after the fire, but it is no more than guesswork to postulate that exposure to the smoke might have accelerated the process. Although Mr Clearkin did not advance the argument in this manner, I accept that it is possible that smoke exacerbated eye symptoms in previously vulnerable individuals, including those suffering from meibomian gland dysfunction, during the course of exposure to it and a recovery period thereafter. Whether the exposure to this smoke plume caused such an exacerbation is entirely case-specific and must be resolved on all the available evidence.

237. To my mind, the following matters of general application arising out of the clinical evidence from the eight experts I heard may be made at this stage. First, in all four disciplines the experts spoke of a gradation between mild irritation on the one end of the spectrum and severe inflammation at the other. Issues plainly arise in the grey area in the middle, but generally speaking I consider that it is valid to think in terms of

irritation being transient, self-limiting and a normal physiological response, and inflammation being pathological and, albeit in all these cases non-permanent, probably over the line and into the realm of personal injury and actionable damage. This interlaces with my analysis of actionable damage in my section on the "Governing Law" (see paragraphs 178-181 above). Secondly, I accept Dr Hind's evidence, read in conjunction with my interpretation of Professor Hay's, that the lowest health thresholds for a chemical such as acrolein are probably designed to indicate the sort of concentrations where certain individuals may be expected to experience an irritant, rather than an inflammatory response. Thirdly, and as a heavy caveat on the previous point, it should continue to be recognised that there is considerable variability, vulnerability and range of tolerance thresholds over any given population. Fourthly, it is common ground between the experts that timing of onset of symptoms matters. In my judgment, the irritant response will be immediate, and in relation to an incident of this nature most people will be likely to know within a short space of time what is causing that response. It is the Claimants' case, after all, that the smoke had a nasty, pungent smell. On the other hand, I agree with Mr Redfern that one might expect considerable variability as regards the timing of onset and offset of any inflammatory response. Finally, and to reiterate a previous conclusion of mine, I do not accept Mr Redfern's submission that the medical evidence, viewed as a whole, supports the possibility of an additive, in the sense of a cumulative, response to sub-threshold exposures. The Claimants can do no better than rely on the trigger values (based on two, consecutive hourly means) and 24-hour means set out in the COMEAP report.

238. There are other generic points too, but I will address these in the final section of this judgment. Perhaps the most important, and controversial, of these is the argument advanced by the Claimants' experts and not the Defendant's that it is appropriate to draw inferences as to causation from the patterns they believed they discerned as to a common, time-associated constellation of symptoms.

THE TEST CLAIMANTS

239. Pursuant to CPR Part 19.13(b) and my order of 5th May 2015 (varying the previous order of 17th June 2014), I am trying 20 Test claims within the overall framework of the GLO issues. The parties originally selected 40 Test Claimants (20 a piece) with the objective of examining the broad range of geographical and personal circumstances across the whole group. I doubt whether the parties' selections were wholly dispassionate and objective, because it must be obvious that the Claimants will have alighted on what they hoped were their best cases, and *vice versa*. Whittling down this sub-cohort from 40 to 20 (with the parties now choosing 10 a piece), in line with proportionate case management and the overriding objective, has enabled me to examine what must be a broadly representative cross-section of the group as a whole. However, I do not make the error of supposing that this sample of 20 would be regarded as statistically significant by any epidemiologist. It is another example of the pragmatism of the common law in operation.

240. My evaluation of the Test Claimants must be both inductive and deductive. Inductive in the sense that their individual features must be scrutinised with care before seeking to draw any more general inferences from them; deductive in the sense that the

generic evidence I have already examined may be capable of throwing light on their individual cases. In a case of this sort, the court must move rapidly from the particular to the general, to the general to the particular, and then back again.

241. Appendix 1 to this judgment comprises a list of the 20 Test Claimants (with information as their post-codes and straight line distance from the plant) and a map marking their locations with reference to the numbers the parties have allocated them.

242. Evidence bearing on the individual Test Claimants is derived from a number of sources. First, they were asked to complete questionnaires as part of the litigation process. Most of these were completed in the first half of 2013, already well over a year after the incident. Secondly, each Claimant has provided a witness statement, and in some instances a supplementary witness statement, in line with court orders. The vast majority of these statements were signed in September 2014. Thirdly, there is the evidence of what each Claimant told the examining clinicians as recorded by them. Finally, but only in some cases, there are relevant contemporaneous GP records. All this evidence needs to be compared and evaluated in this case, assessed as appropriate against the oral evidence given from the witness box.

243. I will examine the Test Claimants in the order in which they gave evidence before me. I need to explain my approach. At this stage I will be doing so without express reference to the plume modelling evidence. This is to avoid the potentially mechanistic thinking which underlay the Defendant's clinical experts' approach, namely that because any given Test Claimant was insufficiently exposed it must follow that s/he could not have suffered any symptoms from exposure. I believe that I have already made clear that a more fluid, less rigidly "scientific", approach is required (at least at this stage in my decision-making), although I certainly should not be understood as saying that the plume modelling evidence can be ignored or circumvented. I will be returning to it at a later stage of my analysis. In assessing the Test Claimants, the focus will be primarily on the individual features of their cases, but I will be keeping an eye on a possible "bigger picture" and identification of patterns and constellations. However, this "bigger picture" cannot drive my inquiry, since that would be to commit the logical fallacy of assuming what needs to be proved. I have already said that I will be taking on board all the available evidence, but an issue does arise as to the weight to be given to expert clinical opinion on the Claimants' side that, generally speaking, the Test Claimants were honest individuals who gave reliable accounts. Dr Swift and Dr August spoke in different terms but to the same effect of intuitive judgments forged from lengthy experience in history-taking and clinical examinations. The Defendant's experts, on the other hand, deferred to me on these matters.

244. I have thought very carefully about this last point, but I cannot agree with Mr Redfern that I should be affording "significant weight" to these expressions of opinion. It is striking in this case that by the stage any Test Claimant was seen by the experts in this case, there were no relevant signs to witness, and no enduring symptomatology. Accordingly, there was no possibility of correlating claimed symptoms with any meaningful clinical examination. It follows that the clinical experts were really in no better position than I was to assess matters of individual credibility and reliability, especially in a medico-legal (as distinct from a purely clinical) context. With respect to them, it might well be said that I was in a somewhat better position inasmuch as the Test Claimants' accounts were thoroughly tested in the crucible of the forensic

process. I do not propose to ignore the Claimants' experts' generally favourable impressions; the weight to be accorded to them should, however, be moderate.

245. Another generic point which falls to be addressed at this stage is the absence, in all bar one case, of any contemporaneous medical note either recording a complaint about the fire or linking symptoms to the fire. I do not accept that there is any solid evidence supporting the proposition that some GP surgeries had signs discouraging appointments and encouraging over the counter remedies. Dr Hardy told me about this, but his sources are unclear and I did not hear it directly from any Test Claimant. I do accept Dr August's point that it is understandable that some Claimants might not mention symptoms to their GPs at appointments for other complaints, but I cannot accept that this should be treated as a universal panacea. Nor can it explain a failure to mention the fire in instances where Claimants *were* attending their GPs complaining of what they now say are fire-related symptoms. On the other hand, I do accept and understand the bewildering variability across human nature: some patients are not interested in possible causes and explanations; all they seek is a remedy.

246. As a related matter, a number of the Test Claimants told me that their GP records are either inaccurate or incomplete. I accept this possibility inasmuch as GPs are extremely busy professionals who may not always write down everything that is said, and may occasionally err. I am more inclined to accept the possibility of a record being incomplete than erroneous. If a trained history-taker records "symptoms 1/52", that will almost invariably be his or her best interpretation of the account being given.

247. The final generic point to be made at this stage concerns the approach I should be taking to the obvious delays in this case. Owing to these, and to the fallibility of human recollection, it is inevitable that witnesses may be unreliable historians and unable to give me more than a general sense of what happened. Some allowances need to be made for this, but not to the point of excess. The burden of proof remains on the Claimants, and they need to satisfy me that they suffered the symptoms they claimed. In very many cases, apart from recourse to the wider picture of apparently similar complaints, the Claimants have nothing else to go on apart from their own evidence.

Mr Gary Mangan

248. Mr Mangan was born on 5th April 1985 and at the material time lived 0.53km from the Sonae plant. In 2011 he was working as a retail supervisor, but he is now employed in a very responsible role as a submarine engineer in the Royal Navy. Mr Mangan does not smoke but he had a history of upper respiratory tract infections, including one incident of shortness of breath in 2008. At about 16:00 on Friday 10th June he went home and could see black or dark grey smoke from the factory. There was an unpleasant burning smell and a lot of smoke. Mr Mangan went running on the Friday evening, and again on the Saturday, modifying his route on account of the smoke plume. He felt a bit chesty when running, and his chest was *"quite tight"* that Saturday evening (he denied any symptoms on the Friday). He did not go running on the Sunday, but remained at home all day. On Monday he went to his GP, without a prior appointment, where he was diagnosed with an upper respiratory tract infection for which antibiotics were prescribed. He had symptoms of a tight chest, a cough, and

phlegm. These symptoms, including those of low energy and disturbed sleep, lasted for about 10 weeks, and he remembered returning to his doctor some weeks later.

249. Under cross-examination, Mr Mangan said that he had no difficulty obtaining an off-the-street consultation with his GP. Paragraph 9 of his witness statement, which stated that he *"decided to make an appointment to see his GP"*, was incorrect. In my judgment, it is difficult to understand why so elementary a mistake was made, if it was indeed made. The GP noted the presence of ulcers on the right side of the palate by the right tonsil. Mr Mangan explained that he did not mention the smoke to his GP on this occasion because he just attended *"for my health"*, and obviously everyone was aware in the area what was going on. He had made the connection between his symptoms and the fire.

250. The GP records show that he returned to the practice on 16th August 2011 for a night cough. He made no mention of the fire. His explanation for this omission was that *"I go to the doctors for my health"*. I understood that to mean that Mr Mangan is not concerned with the underlying reasons for any ill-health, but just practical solutions.

251. Dr Hind felt that Mr Mangan is likely to have suffered a coincidental upper respiratory tract infection. Dr Hardy, taking into account the absence of a temperature, the ineffectiveness of the antibiotics, and the fact that an ulcerated palate is not commonly associated with infection, originally concluded on the balance of probabilities that this Claimant did suffer from the effects of smoke inhalation. However, in cross-examination it was pointed out to Dr Hardy that Mr Mangan claimed to develop symptoms before 12th June (i.e. before the date of any significant exposure to the smoke), and Mr Kent pressed him on the GP record. Dr Hardy then said, *"possibly he had an infection; it is likely at that stage he had an infection"*. He agreed that in his report he could equally have said that Mr Mangan's account was consistent with smoke exposure.

252. In my judgment, Mr Mangan was no better than a reasonable witness who, like many others, could not remember much of the fine detail of what happened. In such circumstances, I cannot conclude that his account was particularly reliable. Not without some hesitation, I do not find that on Friday 10th June he had already booked an appointment to see his GP on the following Monday (had I made such a finding, it would ineluctably have followed that he was suffering from an upper respiratory tract infection before he was aware of any of the effects of smoke inhalation). Even so, on the balance of probabilities I conclude that Mr Mangan was probably suffering from a coincidental throat infection. His symptoms started before there was any significant exposure to the smoke plume, and Dr Hardy agreed in cross-examination that it was likely that this Claimant had an infection.

253. In any event, I am not satisfied that Mr Mangan's symptoms lasted for anything like as long as 10 weeks. If, as a fit young man he was concerned to see his GP for what he believed to be a mere throaty cold, suggesting a low complaint threshold, and had it not gone away after the usual week or so, I regard it as inconceivable that he would not have arranged to see his GP whilst still apparently suffering symptoms. Furthermore, when he did see his GP on 16th August, there was no mention of the fire.

254. I will set out my final conclusions in relation to Mr Mangan's case only after considering all the plume modelling evidence in conjunction with the evidence of the other Test Claimants.

Mrs Tracey Beatham

255. Mrs Beatham was born on 21st August 1972 and lives 1.96km from the Sonae plant. She works as a domestic at hospital, in proximity to cancer patients, and the nature of her employment means that she has to be particularly cautious in not bringing ordinary colds and infections into her work environment.

256. Mrs Beatham is a moderate smoker and in February 2010 was suffering from a persistent cough.

257. According to paragraph 5 of her witness statement:

> "I was not aware of the fire until the following morning when I got up to get ready for work. I noticed a terrible smell of burning. When I looked out of the window, I could see that the whole area was covered with smoke. In particular, I noticed that my garden was covered in dust and ash. The smoke was extremely thick and dark and was blowing towards my house and into my estate. I cannot recall the plume being particularly high at the time of seeing it."

This was at about 06:15, when the wind was blowing eastwards from the plant. Mrs Beatham also noticed considerable quantities of ash and dust which lasted for weeks, as did the pungent smell. She could not hang her washing outside. Many other Claimants gave evidence to similar effect.

258. Mrs Beatham complained of symptoms including a severe cough and chest tightness which lasted for approximately 8 weeks in all. She saw her GP on 24th June, and the following medical record has been retained:

> "Upper Respiratory Tract Infection NOS 1/52 tired, sore throat, cough, running nose. o/e throat red nil pus chest clear advice given. Delayed script"

259. Mrs Beatham has given inconsistent accounts of when her symptoms started. The natural interpretation of the GP note is that her symptoms commenced at around 17th June. According to her questionnaire, they developed within two weeks. Her history as given to Dr Hardy was *"2 days after the onset she developed cough and phlegm so much that she vomited after coughing the phlegm"*. According to her witness statement, they started within the first week of the fire. In the witness box, Mrs Beatham said that her cough started on the third day, and that after about a week she took time off work. She had to wait 3-4 days for the doctor's appointment.

260. In her oral evidence, Mrs Beatham said that she linked her chest symptoms to the fire straight away, as soon as she had her cough (i.e. at around the 2 day point). She said in evidence that she asked her GP if her cough could be related to the fire, but he did

not answer. Her witness statement had said that she could not recall mentioning the fire to her GP at the time. This is consistent with what she told Dr Hind, namely that she assumed that she had picked something up. She could not explain this discrepancy.

261. Under cross-examination, Mrs Beatham said that she also had eye symptoms which she mentioned to her GP. Her questionnaire and witness statement had made no such claim. She could not explain this inconsistency. When asked about the GP note, Mrs Beatham denied that her chest was clear. When it was put to her that she told Dr Hind that *"she was not aware of dust inside the house"*, she denied that she was exaggerating the position.

262. Dr Hardy accepted in evidence that if the symptoms did not begin until one week after the fire, as the GP note recorded and other evidence suggested, then it is less likely that her symptoms were caused by the smoke: the more probable explanation is viral infection.

263. In my judgment, Mrs Beatham was an unreliable witness whose oral evidence could not be accommodated within the far more reliable documentary record. Her account, particularly in relation to the extent of the accumulation of dust and the duration of any symptoms, contains elements of subconscious exaggeration. I express the matter in those terms because I do not believe that Mrs Beatham was intent on deliberately misleading the court, and that in any event has (wisely) not been suggested. Mrs Beatham is, however, a good example of a suggestible witness who could not remember what actually happened, and therefore tended to say what she assumed could be right because that fitted into her mindset of what this fire must have caused.

264. In my judgment, Mrs Beatham's case cannot succeed on the basis of her own testimony. I accept that the unsatisfactory nature of her evidence does not exclude the possibility that she may have suffered from relatively minor symptoms hovering at the threshold of legal actionability, but (at least as regards her own evidence viewed in isolation) she has failed to discharge the burden of proof which remains on her. Unless the plume modelling evidence is supportive and/or she is able to rely on a powerful intra-cohort "constellation of symptoms" effect, her claim should fail.

Ms Jessica Alexander

265. Ms Alexander was born on 17th October 1993 and was only 17 at the time of the fire. She was studying at a sixth form college which was 1.42km away from the Sonae plant. Her home is 1.56km away from it.

266. Ms Alexander is a non-smoker who had pre-existing eczema, including a flare-up over her elbows and knees in January 2011. According to her witness statement, but not her questionnaire, she had previously suffered from styes in or near her eyelids which could cause swelling and streaming.

267. According to Ms Alexander's questionnaire completed on 22nd February 2013, she suffered a range of symptoms, all of immediate onset and floridly described. Her eyes were sore and dry, she was constantly rubbing them, and although her symptoms

resolved within two weeks, she believes that her myopia was brought on by this experience. She developed red and itchy patches across her torso and face, which made her self-conscious at school and caused problems with sleep. After two months, her GP diagnosed contact dermatitis, and after treatment it took a further two months for symptoms to resolve. She developed migrainous headaches which interfered with her exams, and rendered her unable to attend her maths A level exam (this must be a typographical error: according to her witness statement, this was a GCSE maths re-sit). These acute symptoms lasted for around 2 weeks. She also experienced dizzy spells, an increased temperature, and a sore throat.

268. In her witness statement, Ms Alexander stated that there was a lot of dust and ash in the area at the time of the fire. It was sufficiently tenacious to seep into the house, and it was a *"constant job"* to keep on top of it.

269. Ms Alexander was neither a confident nor a reliable witness. In my judgment, she was self-conscious about her appearance before the fire, as many young women are, and it is difficult to link cause with effect. Her myopia could not have had anything to do with the fire, and it became clear from contemporaneous, pre-fire documents that Ms Alexander was experiencing eye problems which were either due to a fly entering her left eye, causing swelling, and/or pre-existing lid margin problems – a feature of meibomian gland dysfunction. Mr Clearkin agreed in cross-examination that it could have been either, but appeared to favour the manifestation of symptoms from the pre-existing condition.

270. Although Ms Alexander was a regular attendee at her GP, she did not make an appointment to see him after the fire. Indeed, when she saw him on 14th June she made no mention of the fire, explaining to me that she was self-medicating and that *"everyone knew about the fire"*. Reading through her substantial, pre-fire medical history, I cannot accept that explanation – it would only make sense if she had not associated her symptoms with the fire at all, which was not her case. She saw her GP again on 8th July and he gave her a sickness certificate covering the period 4th – 11th July, presumably to excuse her from attendance at college. However, by then the exam season had concluded. On 16th August her GP diagnosed "contact dermatitis", and it is surprising that on this occasion Ms Alexander did not mention the fire if she believed, as she told me, that it had caused it. Dr August agreed with the proposition that the itchy patches across the torso described by Ms Alexander are difficult to explain because more naturally exposed parts of the body would receive greater toxicological insult.

271. There is no medical evidence supporting Ms Alexander's other claimed symptoms. Given her unreliability as a witness, I do not accept that she has proved her case in these respects. Her elevated temperature, for example, is highly unlikely to have had a toxicological origin. Her eye and skin problems were pre-existing, and in her case there is nothing to cause me to wish to depart from the advice of Professor Hay regarding the absence of evidence linking dermatological symptoms to smoke. I cannot exclude the possibility that smoke exposure exacerbated Ms Alexander's pre-existing eye condition, but the resolution of that issue depends mainly on the plume modelling evidence. Otherwise, Ms Alexander's case is in the same category as Mrs Beatham's.

Ms Kelly Colebourne

272. Ms Colebourne was born on 29th October 1979 and lived 2.16km from the Sonae plant. She continues to work as a fitness instructor. She remains a non-smoker, and save for one issue relating to possible pre-existing asthma, she enjoyed very good health.

273. Ms Colebourne told me that she awoke at about 05:40 on Friday 10th June to see a thick black cloud of smoke crossing the clear blue sky. By 14:45, when she returned from work, it was *"just smoggy, foggy, cloudy – like smog"*. The smokiness and dustiness continued that weekend, and Ms Colebourne described a *"char-grilled smell, acid-y at the back of the throat"*. She said that her breathing felt a bit funny. After about one week of the fire, according to her questionnaire, Ms Colebourne experienced severe respiratory symptoms, and a deep and chesty cough, which was very uncomfortable. She also suffered sore, itchy and runny eyes, which became red and bloodshot. Her eye symptoms lasted for about 4 weeks. These symptoms also caused low energy and a general feeling of being increasingly worn down.

274. On 5th July 2011 Ms Colebourne suffered a severe asthma attack which resulted in her being hospitalised.

275. Ms Colebourne was cross-examined closely in relation to the timing of the onset of her asthma symptoms. According to her questionnaire:

> "I was absolutely fine before the fire, and only started to suffer these horrible symptoms after it started. As I mentioned above, I had no history of asthma at all before this, and I was not exposed to any other irritants that could have induced it, so it seems obvious that the fire was to blame. "

276. In fact, an examination of Ms Colebourne's medical record reveals that she was complaining of general tiredness symptoms in July 2010, of acute tracheitis in November 2010, and that asthma was diagnosed in February 2011 – in the context of complaints of wheeziness and general malaise. Ms Colebourne informed me that this diagnosis was incorrect, notwithstanding that she was given an inhaler.

277. On 15th June 2011 Ms Colebourne saw her GP, was diagnosed with hay fever, and was advised to take her inhaler. It did not enter her head, she said, to mention the fire to her GP, notwithstanding (a) the terms of her witness statement, to the effect that it seems obvious that the fire was to blame, and (b) the apparent need to contradict the GP's diagnosis if there appeared to be some more plausible explanation for her symptoms.

278. On 5th July the history as recorded by the OOH service was as follows:

> "… since yesterday has had problems with chest since November, not formerly diagnosed with asthma but given a ventolin inhaler. Non smoker. Since yesterday has been coughing phlegm and struggling to clear chest. Feels SOB and has used ventolin 4 + times today. On the phone able to speak in long full sentences but sounds congested …"

279. I read the *"since yesterday"* as a reference to the more serious symptoms Ms Colebourne was obviously experiencing. However, it rather excludes the sort of serious respiratory problems, dating back to one fire after the fire, mentioned in her questionnaire. I also mention the hospital record dated 6th July which states that *"over the past several months has been suffering from increased shortness of breath"*.

280. I was not impressed by Dr Hardy's evidence in relation to this Claimant. He attributed her symptoms to the fire because of the temporal association, and because she had a life-threatening attack after the fire. However, even a cursory examination of the contemporaneous medical records does not bear this out. I much prefer Dr Hind's evidence on this issue. Nor can I remotely accept Dr Hardy's attempt to attribute six months' of symptoms to smoke inhalation.

281. In my judgment, Ms Colebourne was not a dishonest witness but she has persuaded herself into believing, some considerable time after the relevant events, that the fire caused her asthma attack. Ms Colebourne told me that Camps (or their agents) knocked on her door to recruit her to this litigation, and it is also noteworthy that her questionnaire contains serious errors relating to her *"very physical life"* which, to be fair to her, she disowned.

282. It is possible that Ms Colebourne experienced respiratory problems between 10th June and 5th July which were due, at least in part, to the fire, but the only way that she can prove that she did is with regard to the plume modelling evidence and/or the possible constellation of symptoms effect.

Ms Dawn Bunting

283. Ms Bunting was born on 13th January 1974 and lives 0.44km from the Sonae plant. As the crow flies, she is the closest of all the Claimants to the original source of the smoke plume. However, that does not mean that her exposures were likely to have been the greatest.

284. Ms Bunting is a moderate smoker and in the past suffered from rosacea at times of stress.

285. Her case is complicated, if not bedevilled, by the fact that she has completed two questionnaires containing different information. In her first questionnaire, she stated that she first developed a sore throat, sore eyes and a headache within two days of the fire, and that these symptoms resolved within three to four months. She denied that she smoked or had any pre-existing skin condition. In her oral evidence, Ms Bunting agreed that the questionnaire was *"highly inaccurate"*, but a genuine mistake on her part. In her second questionnaire, completed about two months later, she stated that she developed symptoms within 24 hours of the fire, and that her respiratory symptoms lasted two months, her eye symptoms one week, and her skin problems three weeks. Her witness statement times the onset of symptoms at "within 48 hours".

286. There were thick layers of dust or ash inside and around the house. This was really bad within the first week, and took about one month to settle down.

287. There were numerous inconsistencies in Ms Bunting's evidence, which in my view were not satisfactorily explained. According to her first questionnaire, *"I did only think it was a passing flu at first"*. However, in her oral evidence she said that she associated her symptoms with the fire straight away, and her sons had the same symptoms. When her attention was then drawn to the questionnaire, she said that it was a long time ago, and *"maybe I did think it was a passing flu at the time"*. In my judgment, this is the more likely explanation, particularly when it is noted that Ms Bunting attended her GP on 20th June 2011. She went to see him for a long-term problem, but sought to persuade me that she did tell her GP about her cough and sore eyes. She could not explain why this was not recorded. She did accept that she could not recall if she attributed these problems to the fire. I cannot accept that there was any complaint to the GP on 20th June. Ms Bunting's witness statement makes no mention of it.

288. Ms Bunting was recruited to this group when she was out shopping and saw a sign *"were you affected by the fire?"* In my view, she was another Claimant who has permitted herself to embrace a narrative which gained full currency long after the event. I am not to be understood as finding that her solicitors have created a false account; I am making the more parsimonious point that certain individuals are vulnerable, compliant and suggestible.

289. Dr August was asked about the opinions expressed in his report:

> "**Past History** Rosacea. This has been present for the last 5-6 years beginning in 2008-2009 when it was severe for 3 months. This became worse after the fire, particularly on the cheeks and around the eyes where the skin became lumpy and cracked. This seemed to be bad for about 2 months and then settled back to its pre-fire severity.
>
> …
>
> **Opinion** She seems to have had redding [sic] of the face which was worse than before. The rash was certainly confined to the face only. The overall impression is that of rosacea exacerbated by the fire for a period of 3 weeks, possibly longer."

290. Dr August agreed that the evidence in Ms Bunting's case was *"complicated"* because she has not given a consistent account. On any view, her skin problems were not mentioned to her GP on 20th June. There is sparse evidence linking dermatological conditions to smoke exposure, but in any event I am not satisfied on her oral evidence that the case is made out.

291. Hers is another case which is not necessarily doomed to fail on account of her unreliable testimony, but requires a solid basis on the plume modelling and/or a constellation of symptoms effect to stand any realistic chance of succeeding.

Mr Terence Dunn

292. Mr Terence Dunn was born on 12th January 1948 and lives 2.31km from the Sonae plant. He is retired, and in June 2011 went out for substantial morning runs.

293. Mr Dunn had no pre-existing health history of note. He gave up his modest smoking habit in 2006 or thereabouts.

294. According to his questionnaire, Mr Dunn suffered from the immediate onset of respiratory, eye, nose and throat symptoms, together with headaches, dizziness and stomach pain. More specifically, he had a nasty chesty cough, producing shortness of breath, mainly on exertion. This prevented him from going running. His eyes were sore and stinging, and he suffered from what he told me was *"extreme discomfort"*. His nose was very blocked and he had an *"incredibly sore"* throat.

295. On the first morning of the fire, Mr Dunn told me that he was on his usual 6½ mile run but could only do about half. He could not breathe; there was coughing, spluttering and his eyes were watering. Mr Dunn described a *"real putrid, horrible smell"*. First of all, he thought it was akin to a pig farm (he might have been smelling the ammonia in the wood); it left an acid-y biting taste in the mouth.

296. The quantities of dust and ash were such that Mrs Dunn insisted that their vertical blinds be replaced. He paid cash - £200-300. The manner in which Mr Dunn's evidence was given was convincing and credible.

297. Overall, Mr Dunn appeared to give his evidence in a moderate and understated manner. He said that his nose symptoms resolved within one week, and the remaining symptoms within a further two weeks. He self-medicated for these.

298. Provisionally impressed as I was by Mr Dunn's reliability as a witness, I asked him to describe the colour of the smoke plume to me in more detail. He said this:

> "From the first time I saw, it was black, very black.
>
> MR JUSTICE JAY: Very black, yes.
>
> A. And then over a period, it would go blackish grey, if you was looking over that way, but we didn't tend to a lot, and then it would sometimes go a bit white. That was the colours I seen when I looked.
>
> MR JUSTICE JAY: After about a week or so, was it still going black?
>
> A. No.
>
> MR JUSTICE JAY: Was it –
>
> A. It had changed colour, my Lord, after a week. It wasn't black anymore.
>
> MR JUSTICE JAY: In terms of the quantities of smoke, can you give me an idea of that?

> A. First few days, my Lord, was really bad. Really bad, and then it just started to settle down. Just keep -- you know, there was smoke coming from it all the time, rising, but never as serious as the first few days, in my opinion.
>
> MR JUSTICE JAY: Yes. I know it's difficult to describe, but after the first few days, just give me a picture of the quantities of smoke.
>
> A. It's very difficult to, you know -- because, as you say, it had changed colour. So it was not as though you could look over and see something black all the time. You know, you'd notice all the time, wouldn't you? But there were a change in colour. Sometimes it would go very easy and then sometimes it would flare up again and you'd see a white plume or a grey plume. But that's only if you was out looking over all the time."

299. Entirely credible as a witness though Mr Dunn was, this seam of evidence demonstrates how witnesses can be unreliable when it comes to the detail of describing events that happened years before. The memory can pick out the worst features of the event (in some people, the memory can blot these out completely), and in Mr Dunn's case he has unwittingly prolonged the period of the black smoke well beyond its proper confines of the afternoon of Friday 10th June – as vouched by all the expert and photographic evidence.

300. The same unwitting process of prolongation and exaggeration may have impacted on Mr Dunn's evidence regarding his physical symptoms, and the ash and dust. That evidence needs to be balanced against the plume modelling evidence in his case. I will be examining this with care before expressing any further conclusions about him.

Ms Julie Carney

301. Ms Carney was born on 9th March 1977 and lives with her five children (all of whom are also Claimants) 1.94km from the Sonae plant.

302. She is a light smoker who had pre-existing dermatitis, on and off, from the early 2000s, and asthma-like breathing problems for which she had been receiving no treatment.

303. According to Ms Carney's questionnaire, she suffered from a range of respiratory, skin and nose problems *"soon after the fire started"*. These problems are ongoing. She did not link her symptoms to the fire until early 2013.

304. Ms Carney's primary complaint related to her dry, scaly skin – according to her questionnaire, *"most noticeable on backs, arms and torso"*. Plainly, these were the covered areas of the body where one would least expect to see evidence of contact-related skin problems, setting aside the causation difficulties that she in any event faced as regards the dermatology.

305. Ms Carney was taken in cross-examination to her GP records which showed that she was complaining of similar skin problems before the fire. These may have been partly stress-related. She tried to persuade me that the spots on her skin had completely cleared by the time the fire started, but I simply cannot accept her evidence in this regard. Paragraph 24 of her witness statement said *"I have not suffered with my skin since 2009 prior to the fire"*.

306. The following passage taken from Ms Carney's expert, patient cross-examination by Mr Michael Jones serves to demonstrate the overall poor quality of her evidence:

> "Can you explain why these very serious symptoms are described in this witness statement, when it seems that's not your recollection of the symptoms that you have?
>
> A. It's human error, isn't it? It's a misprint on that -- that bit. I'd said -- when I've described my symptoms to the lady who was writing it down, I said my nose was congested and my chest felt heavy and I found it difficult to breathe.
>
> Q. When you were answering questions from my learned friend Mr Redfern, he asked you whether you went to your GP and you said you didn't, and the reason you wouldn't go to your GP was because you had a little cough. You wouldn't trouble a GP with a little cough.
>
> A. I don't trouble the GP with anything.
>
> Q. I haven't asked you the question yet. What you're describing in this witness statement could not fairly be described as a little cough, could it?
>
> A. I just got on with it. I've got five kids. I'm on my own looking after them. So I haven't got time to sit in the doctor's to be told: it's stress and I can't give you antibiotics."

307. Ms Carney went to see her GP on 13th September 2011. She was advised that her rash and cough were down to stress. Ms Carney now feels that they are down to both. However, she agreed in cross-examination she had not made the link with the fire. Given the smell that she described (akin to a hamster cage – not implausible in itself), and the quantities of *"billowing"* smoke that she witnessed, it is very difficult to accept that she did not make the connection – on the premise, that is, that her descriptions are correct.

308. Unfortunately for Ms Carney and for others, her cross-examination concluded on an extremely damaging series of notes:

> "Q. What I suggest, Ms Carney, is that you have no idea if any symptoms you have had since June 2011 are related to this fire, do you?
>
> A. I'm not a specialist, am I?

Q. No. So you've no idea?

A. No, because the doctor hasn't turned round to me and said: this is all down to the Sonae factory fire. He said it's down to stress. But in the area people have got the exact same symptoms and had the same problems.

Q. So you think because other people might have symptoms that they say are caused, you say yours might be caused?

A. There's got to be a link somewhere, hasn't there?

Q. In fact, in 2013, when you went to that shopping centre in Kirkby, you jumped on a passing bandwagon, didn't you?

A. No, I was asked a question and I answered.

Q. What was the question?

A. If I'd suffered anything -- any of these conditions. It was chest, any chest symptoms from the fire, and I said yes, I had. And then they said, would you like to come along and talk to us, and I said yes, because I don't want something like that in the area where my kids are. My kids have got to live here, haven't they? It's not jumping on a bandwagon. It's looking after your family, isn't it?"

Ms Karen Court

309. Ms Court was born on 23rd July 1964 and lives 2.27km from the Sonae plant. She now works in a responsible position as a volunteer care co-ordinator. At the time of the fire she was a training manager.

310. Ms Court completed, and apparently signed, two separate questionnaires. I deploy that adverb because her two signatures look rather different, but she assured me that they were both hers. I have to accept her evidence in this respect.

311. In her first questionnaire (Tandem Law), Ms Court stated that she had not had the opportunity to review her medical records, but to the best of her recollection and knowledge she first developed serious symptoms *"some two weeks after the fire started"*. These symptoms included chest pain, a sore throat, reflux, itchy eyes, itchy skin and a cough. She only became aware of the causal link when she realised that other people in the area were experiencing similar symptoms. According to this questionnaire, she received medical attention at her GP surgery.

312. In her second questionnaire (Walter Barr) she first suffered symptoms on 9th/10th June and these comprised a chesty cough (*"coughing a lot"*), itchy eyes and itchy skin, and it took three weeks for the eyes and two or three months for the remaining symptoms to recover. Ms Court gave a different smoking history in this questionnaire. She

claimed that the Tandem Law questionnaire was rushed, and that she was happier with the Walter Barr one, but it looks the other way round to me.

313. In her oral evidence, Ms Court described a big white cloud of smoke coming towards her house. The smell was *"a little bit like rubber"*. There were *"loads and loads"* of debris. Like many other witnesses, Ms Court was unable to hang out her washing. Her oral evidence was to the effect that her itchy eyes lasted for four months, and her cough for approximately four months. In my judgment, it was clear that she was exaggerating the length of any symptoms, which calls into question her characterisations of the extent of any ash and dust.

314. As with the previous Test Claimant, Ms Carney, Ms Court unwittingly let slip some very revealing answers during her cross-examination by Mr Jones:

> "No, you asked me, when did you first become aware of something; right? And I remember seeing that and thinking -- first thing I thought, I'll be honest, is we were at risk. That fire means that we were at risk. I'd forgotten about it because no one come to the house and said you need to be screened, you could have been at risk, you might have got respiratory whatever.
>
> Q. You had forgotten about the fire?
>
> A. When I seen that I thought, oh, there's been fault with Sonae. There's a claim. Where there's claim, there's blame. And I thought: hang on a minute, my itchy skin and everything, what have I been exposed to?
>
> Q. So that was the first time you'd made a connection between your itchy skin?
>
> A. Yes.
>
> Q. I think you said that was two years later?
>
> A. Yes.
>
> ….
>
> Q. Let me ask you a narrower question. You'd forgotten about the fire; yes? That's your evidence?
>
> A. Yes.
>
> Q. You're walking through Kirkby centre. You see the unit and it says that Sonae is at fault, I think, is the phrase you used a moment ago. Had you not seen that sort of material saying Sonae is at fault, one, you probably would have forgotten about the fire still.

A. I didn't forget about the fire. I'd forgotten about Sonae itself, if you know what I mean."

315. Ms Court was convinced that she first saw thick smoke after 20:00 on the evening of 9th June. She must be mistaken about that. Notwithstanding her first questionnaire, she claimed that she first experienced symptoms immediately, in other words, that night. Her feeling at the time was that her symptoms would be a temporary inconvenience, but her prolonged dermatitis suggested otherwise.

316. Ms Court saw her GP on 10th and 13th June in relation to other matters. She said that although there was no mention of any symptoms from the fire, she was sure that she did discuss it. The GP's advice was to take Betnovate for the dermatitis. She was convinced that this advice was given on the second occasion. Paragraph 19 of her witness statement claimed that she could recall one occasion on which her cough was so bad that she thought that she would die. According to her oral evidence, this was the immediate impact of the fire, which must have been before 13th June. If that is right, one would have expected the GP to have noted a complaint; and even if Ms Court is wrong and this occasion was after 13th June, it is very surprising that symptoms of this severity did not attract a specific visit for medical advice. I note too that Ms Court disowned paragraph 32 of her witness statement, which claimed that a week after the fire she took her son to the GP because he was unwell, and the GP recommended lotion for her skin.

317. Finally, I should state that Ms Court agreed that her claim in the sum of £500 for additional paint and cleaning material was probably overestimated.

318. In my judgment, Ms Court was an unsatisfactory witness the reliability of whose evidence I cannot accept. Her claim only has a prospect of success if it supported by the plume modelling evidence and/or a constellation of symptoms effect.

Mrs Annette Farrell

319. Mrs Farrell was born on 18th April 1952 and lives 1.81km from the fire. She was an administrative officer at a sports college, and retired in September 2011.

320. She is a light smoker and had pre-existing problems with bronchitis and shingles.

321. According to her questionnaire, Mrs Farrell suffered a range of respiratory, eye and ENT problems following the Sonae fire. These all developed within 48 hours. Her respiratory problems are ongoing; her eye symptoms resolved within one month, her ENT problems within three months.

322. In her oral evidence Mrs Farrell described the colour and quality of the smoke, in particular its horrible, chemical smell – *"a bit like glue"*. She said that it burnt the back of her throat, went inside her nose and made her eyes water. The gritty atmosphere, which was similar to the morning after bonfire night, lasted for two to three days.

323. Mrs Farrell was diagnosed with asthma in September 2011, and in November 2011 went onto the asthma register.

324. In cross-examination, Mrs Farrell said that she had not suffered from breathing problems before the fire. However, a letter from the Aintree Chest Centre to her GP dated 10th November 2010 indicates clearly that she had a wheezy cough and was quite breathless on exertion. The physician wondered if she had bronchial hyper-reactivity.

325. On various occasions in 2010 and 2011, Mrs Farrell was prescribed Salbutamol. On 23rd March 2011 she was prescribed steroids for her chest. According to his report, she told Dr Hind that she used an inhaler approximately once a day during the year before the fire. She could not remember telling him this, nor could she really explain why her questionnaire had denied any asthma symptoms.

326. Mrs Farrell told me that she was sure that she working at the time of the fire. She was taken to a series of medical records showing that she was given sick certificates for shingles between April and August 2011. She said that some of these were backdated, and that she was back at work on 6th June. At the time she was giving evidence, her occupational health records were not available, but now that they have been obtained they clearly show that the sickness certificates were not backdated and that Mrs Farrell was off work with shingles over the whole of this period. I accept that Mrs Farrell was not seeking deliberately to mislead me, and – as her recent witness statement says - that she may have become confused about the exact dates. However, the revelation of these records significantly undermines the reliability of her evidence.

327. Mrs Farrell attended her GP on 23rd June 2011 but there is no record of her mentioning any eye and nose complaints to him. She told me that this was an omission in the GP notes, and that she did; but that answer is difficult to reconcile with the note of a complaint of a wheezy cough, which was in very similar vein to previous complaints. If there had been something new, it is highly likely that the GP would have recorded it. I have taken on board Mr Swift's point, made in the context of Mrs Farrell's case, that when people go to their GP they do not always mention all their symptoms; but her evidence was that she did so on this occasion.

328. Mr Swift also had this to say about Mrs Farrell's case, which I considered to be illuminating:

> "There is also -- it's not necessarily the person that's trying to tell mistruths. It may be their perception of things. So you find that if someone suffers from, say, anxiety and depression, which are quite common disorders, certainly in the rhinology field, their perception of having a stuffy nose will be much greater than someone who is happy with life. I'll quite often sit in clinic, trying to work out why they've come to see me, because my nose is generally much worse than theirs, and that is an honest opinion on that."

329. Overall, Mrs Farrell was not a reliable witness, and her case cannot in my view succeed on the basis of her own testimony. As with other Test Claimants falling within this category, she requires the plume modelling evidence and/or a constellation of symptoms effect to support her.

Mr James Reece

330. Mr Reece was born on 21st January 1953 and lives 3.1km from the Sonae plant. He works as a night janitor at Knowsley business park. He has a history of chest symptoms and hay fever, and is a heavy smoker.

331. According to his questionnaire, he developed symptoms on or about 12th June 2011. He described these as *"a bit of a cough and … cold flu like symptoms"*. He believes that he now has asthma as a result of being exposed. Maybe it was for this reason that he described his symptoms as *"ongoing"*. It was not until he saw an advert in a newspaper that other people had apparently suffered similarly that he made the link between his symptoms and the fire.

332. In his oral evidence, Mr Reece explained that he felt that his symptoms – of what he believed was hay fever – came on a bit stronger. He felt miserable.

333. Under cross-examination, Mr Reece said that he was sure that he saw the fire before 04:00 on 10th June, when he went outside for a smoke during the course of his employment. He must be mistaken about that. He made an appointment to see his GP on 22nd June but did not attend. He told me that he wasn't thinking straight, although the appointment was probably arranged for his symptoms. On 5th December 2011 he attended his GP complaining off a cough which had lasted for two months. The GP records show that asthma was not diagnosed until 19th October 2012.

334. Dr Hardy's report ascribes conjunctivitis and rhinitis to smoke inhalation, and also a severe acute bronchitis which became chronic. Dr Hardy is unclear about the aetiology of Mr Reece's asthma. Dr Hind defers to my view of Mr Reece's credibility and the plume modelling. In my judgment, Mr Reece was an entirely unconvincing witness, and I would be unwilling to attribute any symptoms to smoke inhalation unless the plume modelling evidence and/or a constellation of symptoms effect is available to support him.

Mr Edmund Kenny

335. Mr Kenny was born on 8th December 1951, and lived 1.8km from the Sonae plant. He is retired and for some years cared for his mother, who sadly passed away in February 2015.

336. Unfortunately, Mr Kenny's overall health is not good. He has a heart condition and is diabetic. He was a heavy smoker for many years, suffers from hay fever, and has experienced episodes of shortness of breath. Mr Kenny has also been an asthmatic since 1990, but he told me that he manages to keep it under control.

337. Mr Kenny was at home at the time of the fire. He told me that he was aware of it during the evening of 9th June. By the morning, the smoke was a grey/white colour. At lunchtime, it went black. There was loads of it; it was like the smoke coming off the top of a volcano. Mr Kenny also described an awful, glue-like smell with chemical overtones.

338. Unlike many other witnesses, Mr Kenny has given a consistent account throughout the course of this litigation. In his oral evidence, he said that his breathing *"went terrible"* within a couple of days. His eyes were sore; he had a blocked nose and sore throat; he just felt rotten. Consequently, Mr Kenny had to use his inhaler twice as normal, and he used Optrex 4-5 times a day. His symptoms lasted for approximately three weeks in all.

339. Mr Kenny also told me about *"an awful lot"* of dust and ash in the smoke. It was grey, and got everywhere. It was completely different from *"Sahara sand dust"*. He had to clean and wash more often, and try to keep the windows closed. He placed masking tape on the gap over the double-glazing. A sense of the duration of this phenomenon did not clearly emerge from Mr Kenny's evidence in chief, but he told Dr Hardy that he had to keep the windows and doors closed for 2-3 days. Under cross-examination, he said that the dust etc lasted for at least a week.

340. On 20th June 2011, Mr Kenny saw his GP, Dr Mohammed Khan. The computer records reads:

> "H/O; asthma. Flare up after recent Sonic factory fire. Speaking full sentences. Resp sys – b/l air entry equal. b/l scattered wheeze+. RR-18/min. Was more worse over w/e, it seems. P]-Oral steroids. Continue inhalers …"

341. The Defendant accepts that Mr Kenny suffered an exacerbation of asthma in mid-June 2011. The issue is what caused it. Mr Kenny said in cross-examination that his hay fever tends to be worse in May/June, and then in August/September. He agreed that the general pattern was of *"good and bad days"*. There had been a deterioration in 2010 and Mr Kenny was on inhalers throughout that and the following year. He was taking prednisolone before the fire.

342. Mr Kenny was asked about Dr Khan's note, *"was worse over weekend"*, which he agreed was accurate. He said that it was still musty and foggy over the weekend on 18th/19th June. He was not sure whether there was still smoke and dust. Mr Kenny made no mention to his GP of any nose and eye symptoms. He agreed that his cough was getting better by then.

343. Mr Kenny suffered from a serious bout of food poisoning at the end of June 2011. When hospitalised on 2nd July it was noted that he had no respiratory symptoms.

344. In my judgment, Mr Kenny was an entirely honest witness. He is the sort of person who I would also describe as being reliable, but an issue arises as to the quality of his recollection, as it would in relation to anyone being asked to recall events of this nature occurring some time ago. Thus, if he tells me that the dust lasted for a week, I believe him; but he could simply be mistaken about it.

345. Mr Kenny is convinced that cause and effect has been established in these circumstances, and I fully understand his reasons. A final decision cannot be made in his case without considering the plume modelling evidence.

Mr Paul McLoughlin

346. Mr McLoughlin was born on 26th January 1962, and lives 1.61km from the fire. He is a taxi driver who works school shifts and a number of night shifts. He is a light smoker, and has no pre-existing health history of note.

347. For obvious reasons, given the nature of Mr McLoughlin's work, the plume modelling in his case has been difficult to undertake. It has been tethered to his home address; no other modelling exercise would have been possible. I have to take a sensible view about this. Mr McLoughlin's driving would have taken him over a reasonably broad area; and, on occasion, closer to the Sonae plant than his home. There is no satisfactory evidence as to the extent to which the "boxed" environment of a motor vehicle provides any measure of protection.

348. According to Mr McLoughlin's questionnaire, he developed respiratory, eye and skin symptoms within 48 hours of the onset of the fire, as well as headaches. He complained of a persistent cough, sometimes painful and causing difficulties in sleeping. His eyes felt dry, itchy and sore, and would water when driving such that he had to pull over to the side of the road and apply eye drops. His forearms turned red and itchy, and the skin flaked as he scratched. He had to apply Sudacrem and an anti-bacterial soap. The headaches caused a lot of pain in his forehead and elsewhere. All his symptoms resolved within about two weeks.

349. Mr McLoughlin told me that he was at home when the fire started. He saw grey, dark soot-coloured smoke and experienced the "very unpleasant smell" of burning wood and creosote. He also said that there were substantial quantities of ash and dust, which meant that he and his family had to keep the windows and doors closed.

350. An issue arose in cross-examination as to whether Mr McLoughlin was working on 12th/13th June. The computer record suggests not, but he said that he worked because he had to, and would have used a different computer code. I accept his evidence on this point.

351. On 23rd June 2011 Mr McLoughlin saw his GP. He is not a frequent attendee. The computer record reads as follows:

> "C/O – cough had cough for a few weeks now, has been taking OTC medication, chest seems clear. Advised to continue with meds and come back in 2 weeks if no better."

352. Mr McLoughin said that he did not mention his other symptoms to his GP; he was more concerned about the cough. He said that he thought that the fire was mentioned to his doctor in passing. I do not find that it was, otherwise the other symptoms would surely have been raised.

353. Dr August was cross-examined about *"the main symptoms of pruritus unspecified"* which *"could be inferred as irritant dermatitis (but there is no direct clinical evidence)"* or *"urticaria … or prurigo"*. Dr August agreed that, as a general proposition, one would expect such condition(s) to be more widespread, and not confined to the forearms. However, a lot is not known about the distribution of skin rashes. Further:

> "MR KENT: It sounds as though this is quite difficult retrospectively to diagnose.
>
> DR AUGUST: Yes. It's inspired guesswork, really. It's a hunch, what do you think is the best fit."

354. In my judgment, Mr McLoughlin's skin symptoms require consideration within the ambit of the overall frame of the toxicological evidence in this case. As I have already made clear, that evidence is unsupportive. As for his eye complaints, Mr Clearkin's view is that these lasted just two weeks, but he did not accept that this presentation was more consistent with infection than tear film instability. I have difficulty with Mr Clearkin's logic, and I take Mr Marsh's point that eye problems do not appear to have been mentioned at the GP consultation on 23rd June.

355. I have found Mr McLoughlin to be one of the most difficult witnesses to assess. He came across as a reliable person, pleasant and understated. I have little doubt that some key features of this experience have remained in his mind: the smoke; the unpleasant smell; the quantities of ash; the cough; the itchiness. However, as with other witnesses, I was left without a clear impression of timing, duration and severity.

Mr Peter Shaw

356. Mr Shaw was born on 31st May 1967. He works as a driver and labourer. His depot is the premises of his employer, Kings Construction, which is based on the Knowsley Industrial Estate 0.55km from the Sonae plant.

357. Mr Shaw is a non-smoker who had no chest symptoms of any note for a number of years.

358. Mr Shaw falls into a different category from other witnesses. His workplace is south-east of the Sonae plant. For him, therefore, Saturday 11th June is a potentially important date.

359. Mr Shaw normally works Monday-Fridays. His work pattern was and remains such that he is at the depot for approximately 80-90 minutes a day; otherwise, he is on the road. Mr Shaw arrived at work at approximately 06:50 on Friday 10th June. He saw fire engines and a lot of black/grey smoke. He started to suffer from a cough the day after the fire started and while he was at work. Over the weekend, he began to suffer from itchy eyes. He found it difficult to breathe and his work and sleep were affected.

360. Mr Shaw told Dr Hardy that he was *"probably"* working on Saturday 11th June, and Mr Clearkin that he was working on that date. Since then, his payslips have been obtained, and these contradict his account. His explanation is that he *"may have been mistaken"*. It was put to Mr Shaw that he well knew that his case would be enhanced if he could show that he was exposed to the smoke plume on that date, and that in effect he has sought deliberately to misrepresent the position. Not without some hesitation, I acquit Mr Shaw of that aspersion. He was not a particularly impressive witness, but I do not conclude that he would so brazenly have attempted to mislead.

361. A clear issue arises as to the timing of the onset of Mr Shaw's cough. Under cross-examination he said that his sore throat developed over the Friday afternoon, and the sore eyes, cough and phlegm over the weekend. On 13th June he saw his GP who recorded *"cough one week"* and prescribed amoxicillin. At that stage, Mr Shaw said that he had not made a link in his mind between the smoke and his cough. However, he contested the accuracy of the note. On 20th June he re-attended: he was still *"bunged up"*, and the GP prescribed a short course of steroids. Mr Shaw told me that on that occasion he did not mention the fire. It was only *"maybe a week or two after"* that he began to make that connection.

362. Mr Shaw also said that his cough largely cleared after one week, but he was not totally better until 6-8 weeks later.

363. In my judgment, Mr Shaw's case faces problems whichever way the evidence is interpreted. If the medical record is correct, which in my view it probably is, then his cough was not due to the fire. The possibility that the fire exacerbated his symptoms cannot be excluded, but cannot be substantiated on the balance of probabilities on the basis of Mr Shaw's account alone. If the cough in fact developed on Saturday, when Mr Shaw as at home far away from the scope of the smoke, Dr Hardy conceded that *"it's not due to the smoke, I guess"*.

Mrs Teri O'Brien

364. Mrs O'Brien was born on 25th June 1981, and lives 2.63km from the Sonae plant. Until recently, she worked as an emergency operator for Merseyside Police, and is now a Probation Service officer.

365. Mrs O'Brien is an ex-smoker. She suffers from hay fever, intermittent chest symptoms from smoking-related bronchitis, and possibly late-onset asthma.

366. According to her questionnaire, Mrs O'Brien suffered from a range of eye, respiratory, throat and nasal symptoms, accompanied by headache and fatigue, all starting about 48 hours after the onset of the fire. She had never experienced similar symptoms before. These symptoms all resolved after three weeks. She was working normally throughout this period – she could not take time off, because the Police are sticklers for sickness absences.

367. Mrs O'Brien was asked about the timing of the onset and offset of the smoke and accompanying acrid, chemical smell. She said this:

> "That day it was quite intense, for the first few days leading off, and then obviously towards the end of that week it petered out a bit."

368. In answer to my questions, Mrs O'Brien said that she saw black smoke at around 13:30 on Friday 10th June, when it was close to her house, although she could not say how far away. Given that the wind had been blowing from the west for the past five hours, at the very least Mrs O'Brien's timings must be incorrect. On subsequent days, she said that the atmosphere was smoggy and murky.

369. She also said that she attempted to make an appointment at her GP. She gave similar evidence to other witnesses about dust and not hanging out washing outdoors.

370. Under cross-examination, Mrs O'Brien was asked to be more precise about the severity of her symptoms. Her evidence was that they were more intense during the first week, but began to abate thereafter. Mrs O'Brien said that her symptoms were *"alarming"*, but I agree with Mr Kent that if this were really the case it is difficult to understand why she was unable to fit a doctor's appointment into her part-time work schedule. Her explanation was that the appointments she was offered were inconvenient, and that she had other things to do.

371. In my judgment, Mrs O'Brien was a reasonable witness but, in common with many others, imprecise on key issues such as timing and intensity of symptoms. As I have already observed, the notion that her symptoms were *"intense"* does not tally with her explanation for not seeing her GP. Another difficulty from her perspective is her distance from the Sonae plant at all material times. I will be returning to this issue in the context of the plume modelling evidence.

Mr Francis Glascott

372. Mr Glascott was born on 17th November 1945. He lives 2.1km from the Sonae plant, and is retired. Owing to mental health difficulties, his witness statement was admitted in evidence under the Civil Evidence Act 1968. I also acceded to Mr Redfern's application that a Litigation Friend be appointed.

373. Mr Glascott has a history of chest pain and hypertension. COPD was diagnosed after the fire. He claims that within two weeks of the commencement of the fire, he developed respiratory symptoms which lasted for 10-12 weeks. Inexplicably, Dr Hardy has attributed six months' symptoms to the fire. In Dr Hind's view, the entirety of Mr Glascott's symptoms are ascribable to smoking and undiagnosed COPD.

374. On 30th May 2013 Mr Patrick White of GT Law, Solicitors, wrote a "Final Chaser" to Mr Glascott. He was told that the Statement of Truth to be appended to his questionnaire had to be completed and signed by him immediately, so that it could be returned to the Defendant. He was told that if he failed to comply, there could be costs sanctions against him personally. Aggrieved by the peremptory terms of this letter, Mr Glascott wrote to Clyde & Co (undated, but stamped as received on 11th June 2013) and said this:

> "1. Cold called on doorstep by young man regarding fire at Sonae factory in Kirkby.
>
> 2. Was asked had it affected me and although I said no, I was persuaded to give my details and answer some questions (including health questions).
>
> 3. As I had breathing difficulties (and told him so) I decided to go ahead and sign the, what I thought, the questionnaire.

> 3. Was subsequently phoned by GT Law about signing a statement of truth.
>
> 4. Had been diagnosed that my breathing problem was mild emphysema caused by my long-term smoking so I informed GT Law that I did not wish to proceed and I did not believe that the Sonae fire had any bearing on my current health problem.
>
> 5. Was told that I couldn't withdraw my claim as the 7 day cooling off period from the date of the cold caller visit had expired.
>
> 6. I told them I did not wish to proceed with what would then be a fraudulent claim and was informed that if I withdrew I would have to pay their costs.
>
> 7. Subsequently received enclosed letter reiterating their claim for costs …"

375. The Defendant has now pleaded that Mr Glascott's is a fraudulent claim. It should be noted that, notwithstanding the terms of his letter, he has signed a witness statement and did submit to examination by the respiratory physicians.

376. Mr Patrick White agreed to give evidence in line with his witness statement dated 21st May 2015. He denied that Mr Glascott had been cold-called, and said that the latter would have attended GT Law's Kirkby office. He agreed that GT Law's agents went round to Mr Glascott's home in order to ask him to sign the questionnaire. Mr White told me that when Mr Glascott spoke to him on 14th May 2014, he did not wish to proceed. Mr White accepted that he explained to Mr Glascott that he might suffer a costs penalty if he withdrew. Under the terms of the CFA, it was open to GT Law to claim costs from the client, but Mr White said that it is unlikely that they would have done so. According to the terms of the attendance note of the call, Mr Glascott was warned that if he backed out now, the Defendant *"will seek him for any charges"*. This was notwithstanding the terms of Clyde & Co's letter dated 11th July 2012 under which the Defendant irrevocably undertook not to seek payment of their legal costs. Mr White tried to suggest that it would have been negligent of him not to mention possible costs consequences, but in my judgment the letter could not have been clearer. Mr White denied that he was treating Mr Glascott as a *"cash cow"*. He said that Mr Glascott terminated the call by saying, *"f – off"*.

377. Mr White was asked a close series of questions about Mr Glascott's questionnaire and the signature on the Statement of Truth. There are obvious errors and inconsistencies in it. The signature looks nothing like Mr Glascott's signature elsewhere, including his signature on the letter to Clyde & Co. Mr White maintained that it was Mr Glascott's signature. I am not satisfied that it is.

378. Mr White sought to persuade me that he was acting at all material times in Mr Glascott's best interests. In my judgment, he was a poor, rather self-important witness who was acting in what he thought were the interests of his firm rather than those of his client.

379. I am not satisfied that Mr Glascott is guilty of advancing a fraudulent claim. He cannot be held responsible for the conduct of his solicitors. In order to reach such a conclusion, I would need to know more about the circumstances in which he agreed to sign a witness statement and to submit to medical examination. However, he cannot persuade me by dint of his oral evidence (or indeed Dr Hardy's exorbitant attributions) of any causal connection between the fire and symptoms. He is solely reliant on the plume modelling evidence.

380. This leaves the position of Mr Patrick White and GT Law. Mr White is not a solicitor, but he was acting under the direction of his principals. I direct that a copy of this judgment be sent to the Solicitors Regulation Authority for investigation of the issues raised by Mr Glascott's case.

381. This nugget of evidence not merely leaves an unpleasant miasma of concern and dubiety in relation to Mr Glascott's case, it has the potential to infect the integrity of GT Law's processes overall, and other claims. Whether that potential is achieved in all the circumstances of this GLO will need to be addressed later.

Mr Leon Swift

382. Mr Swift was born on 14th February 1989 and at the time of the fire was a trainee accountant (he is now chartered). His place of work was 0.88km east north-east of the Sonae factory.

383. Mr Swift arrived at work at about 08:45 on Friday 10th June. Whilst driving to work along the East Lancs road he could see a large cloud of pale-coloured smoke. Nearer the office, it was smoggy and foggy. From closer-up, the smoke was in a vast quantity and a lightish/dark grey. The smell was strange and different from normal. It did not appear to impact on his breathing at that point.

384. Later on, while Mr Swift was in his office, he began to suffer from itchy eyes and sinuses. This caused him to sneeze. Everyone was talking about the fire.

385. On Monday 13th June the atmosphere was not as foggy as on the Friday, but there was *"definitely"* still a large cloud above the factory. Although there was less ash on his car, his symptoms had deteriorated. He had a slight headache, and his eye and sinus problems continued. Mr Swift told me that he tends not to visit his GP, and so he self-medicated. He bought stronger anti-histamines than he ordinarily used, but these did not really work. It took him 2-3 months to recover completely.

386. Given other evidence in this case which I will be addressing below, the Defendant has pleaded that this is a fraudulent claim. In these circumstances, it is necessary to examine the evidence Mr Swift gave under cross-examination with particular care.

387. Mr Swift completed the standard questionnaire on 27th August 2013. He stated that his symptoms lasted *"3-6 months"* (cf. his oral evidence), and that he made the connection with the fire only several weeks after the incident, following talking to colleagues. Given that the symptoms were apparently of immediate onset (cf. his oral

evidence, which suggests a short delay) I find that difficult to accept. Mr Swift also accepted in cross-examination that the 3-6 month attribution was *"excessive"*.

388. Mr Swift was taken to an undated and unsigned "Sonae Enquiry Form" which appears to relate to him, and states that his symptoms of headaches and nausea lasted for 4 months. He denied writing it. However, in his supplemental witness statement dated 15th May 2015, Mr Swift said that he *"completed and returned"* this form on 19th March. In the witness box he said he may have chatted to someone over the phone. He tried to persuade me that he did not know what the word "nausea" meant.

389. On or about 22nd March 2013, Mr Swift completed a short questionnaire. In it he stated that *"days after the fire I began to be affected with headaches as well as this I suffered breathing problems/ an irritating cough developed"*. Thus, here appears a somewhat different account. Paragraph 3 of this questionnaire is also relevant:

> "*When did your symptoms reach a stage when you informed your employer or GP?*
>
> Unfortunately I did not as I believed the headaches related to working at a computer. It is only now that it has been brought to my attention that I have linked the symptoms to the fire."

390. Mr Swift told Dr Hardy that he worked on Saturday 11th June. Dr Hind's note of Mr Swift's history is somewhat internally inconsistent. Mr Swift's oral evidence was that he did not work on the Saturday. He believed that his account must have been lost by Dr Hardy in the translation.

391. Mr Swift agreed that in the first week after the fire he played 5-a-side football and went to the gym as usual.

392. In December 2008 and December 2009 Mr Swift was involved in road traffic accidents, and brought claims for compensation which were successfully resolved.

393. On 22nd February 2013, Mr Swift was involved in the following conversation on Twitter:

> "**Leon Swift** either of you's jumped on this sonae claim bandwagon?
>
> **TC** been all over the radio
>
> **MC** residents living close going to solicitors due to harmful emissions from the plant
>
> **MC** looks like everyone's doing it now because it's shut down
>
> **Leon Swift** they've admitted liability so anyone living or working in the area at the time of the fire can claim
>
> **MC** get on it ken/tom
>
> **MC** not for me *#too honest*

> **Leon Swift** too honest ya, good one matt. I'm getting involved I reckon, pays for the summer holiday if it goes thru
>
> **TC** ha ha you're a bad man Leon
>
> **MC** he's a fraud Tom
>
> **Leon Swift** takes a fraud to know a fraud Matthew. Mr 'I was in that car that crashed ye' *#showmethemoney*
>
> **MC** my neck was sore when Dave crashed *#thetruth*
>
> **Leon Swift** Asking for trouble driving in flip flops
>
> **MC** if you crash give us a shout *#whiplashclaim*
>
> …
>
> **MC** I'm sure you was fine that time Dave had a crash
>
> **Leon Swift** least I was in the car though Matthew
>
> **MC** so was I"

394. Mr Swift tried to dig himself out of the massive hole created by these exchanges. He said that the use of the term *"bandwagon"* was not the best choice of words. He agreed that the Tweets could be construed as indicating that any claim he made would be fraudulent. However, he told me that he was not saying at the time that his claim was not genuine. He well understood, he said, that an admission of liability did not mean that one could recover damages regardless of injury.

395. I watched Mr Swift very closely during the course of Mr Jones' well-briefed and well-constructed cross-examination. One possible explanation for his extreme discomfiture and obvious embarrassment was that his Tweets were being taken out of context, and he was ashamed by the impression they may have been making. Another explanation is that he well knew that the Tweets contained accurate insights into his true state of mind. Making allowances as I do for the degree of banter that may accompany much discourse over these social networks, but having regard to all the available evidence, I regret that I have to favour the second explanation.

396. Mr Swift is a well-educated young man and ought to be ashamed of himself. The Defendant's pleaded case of fraud has been proved to the requisite standard. His claim fails.

397. Extremely damagingly not merely for him but potentially for others, is the following exchange with counsel at the end of Mr Swift's cross-examination:

> "Q. If you were able positively to make a link, you would have made that link when the fire was burning, not later?
>
> A. Yes.

Q. You have just said "yes". Are you agreeing with that proposition?

A. No. Like it is difficult to remember exactly when you do make the association of the two. As I said previously, a lot of symptoms that I did experience are very similar symptoms to what I experienced on a regular basis. So it is difficult to differentiate between the two.

Q. Exactly. Mr Swift, what happened is this. You would have great difficulty differentiating between symptoms you had anyway and symptoms from the fire, which is why you didn't make any association in those two months or so that you've said occurred before you made an association.

A. Yes, but that's not to say that there wasn't necessarily an association between the two sets.

Q. That's a separate question. The question is whether you can say there's an association. What I suggest is it was only a very significant period later, whether months or in fact I suggest early 2013, that you saw advertising literature, and you thought: well, I can say that those symptoms -- I'm not sure how they were caused -- were caused by that fire and I can get some money; do you accept that?

A. That may have been the trigger that made me think about it again, but it wasn't the financial thing that I was thinking about in terms of making a claim. It was just I realised that the nuisance it had caused at the time, that I basically wanted to bring the claim.

Q. The nuisance it had caused? What do you mean by the nuisance it had caused?

A. Basically, if you're sitting in an office and you've got itchy eyes and tickly cough and sore nose, it's just -- it's inconvenience that you could do without when you're trying to work, isn't it?

Q. But those are symptoms that are so similar to the symptoms you had anyway at the time of the year each year that you couldn't positively link those symptoms to the smoke, could you?

A. As I said, though, those symptoms were heightened considerably. For example, I take Cetirizine, and that controls my hay fever. Things like that. I don't need to take anything additional during the time of the fire, I had to then go and purchase additional in order to try and appease the symptoms that I was referring to.

> Q. If that was true, Mr Swift, you would have made the link there and then, wouldn't you? You would have thought: why is any normal medication that always resolves my problems not resolving my problems?
>
> A. You would think so, yes."

398. Finally, I note that in his report Dr Hardy fairly stated that Mr Swift developed no chest symptoms whatsoever in consequence of smoke inhalation, but then felt able to say that he probably suffered from conjunctivitis, rhinitis and acute bronchitis (cough) for *"up to six months"*. My only observation is that his sort of expression of opinion discloses the dangers of leading with the chin.

Mr Shaun West

399. Mr West was born on 29th December 1988 and lives with his mother 0.58km from the Sonae plant. In 2011 he worked in Aintree as a sales' advisor.

400. Although a virtual non-smoker, Mr West has a history of skin rash (2001-2009), asthma (since 1999), URTIs (1994-2010), rhinitis, sore throat, eczema and hay fever.

401. At paragraph 17 of his questionnaire, Mr West stated that he experienced respiratory symptoms for a period of less than 2 months, eye symptoms for less than 1 week, skin problems for less than 3 weeks, nasal symptoms for less than 2 months, and generalised dizziness, fatigue and aches and pains for less than 2 months. In the witness box he said that he felt that he had *"short-changed"* some of his symptoms, in particular the eyes.

402. In his oral evidence Mr West said that when he came home from work on the evening of Thursday 9th June he was aware of the fire almost immediately. He had a clear view from the hallway window of his home. Between 20:00 and 21:00 that evening, he could see flames and smoke; he was very confident of that (his confidence was misplaced). The next day, in the morning, the fire was really bad and the smoke was starting to billow. It was relentless, and a very dark colour. He could smell and taste it - Mr West, in common with others, described a "chemically" smell. By the time he returned home at around 16:00 to 17:00, the smoke *"still looked incredibly bad"*. Once he was in the house, he shut all the windows.

403. Mr West said that it was very likely that he remained at home that weekend. There was no real respite from the smoke. He figured that everyone was in the same position, and that his GP would not be able to change his environment.

404. In terms of the evolution of the smoke plume, Mr West said this:

> "But I'd say a couple of days in, after the immediate fire getting very severe, there was one or two days when it would be a lot worse in terms of maybe the smoke being lower. The house, I was always surrounded by it, and to be honest, I constantly had the curtain shut."

and subsequently:

> "Q. One final question. What was the atmosphere outside the house like in the period of the fire and the period immediately following it? The air quality, if you like.
>
> A. Very poor. It felt like I was trapped in the house. Any instance where I would have normally walked to the shop to get, you know, even bread and milk, I would have drove to a garage in (inaudible) because I didn't really want to go walking down the road.
>
> Q. That's what you did as a result of it, but what was the air quality like? Can you describe it?
>
> A. I would cough immediately.
>
> Q. That's a consequence.
>
> A. A consequence.
>
> Q. What was the air quality like?
>
> A. Okay. Sorry, could you rephrase that?
>
> Q. Yes. When you walked outside, was it a nice clear day or what?
>
> A. It obviously got worse on some days. So some days I couldn't see across the fields because of the direction the smoke was going in and I live very close to the fields. It was constantly smelling of the smoke and the fire and whatever else. God knows what was in there. But it was pretty horrid, to be honest."

405. In this sequence of evidence, Mr West was certainly not guilty of short-changing himself or the Claimants generally. Being much closer to the Sonae plant than almost every other Claimant, he was well-placed to describe the direct and obvious consequences of the fire. However, his account bears no sensible relation to all the objective evidence in the case, in particular the expert evidence of Dr Mitcheson, the lay evidence of Dr Jowett and Mr Whitrow (in particular), and the photographs which were separately provided to me by the Defendant on a memory stick, including the photograph I mentioned at paragraph 76 above. Unwittingly or otherwise, Mr West's account was heavily freighted with hyperbole.

406. Mr West was cross-examined closely about his claim that there were considerable quantities of dust, ash and debris on the field between his property and the plant. He said in his questionnaire that the field was covered in dust and debris, and he was unable to take his usual walk. The photographs do not bear him out at all. Under cross-examination Mr West made clear that he did not examine the field in any detail, because it was not a good place to be. He later said that there was bound to be dust and debris after a fire, but could not support that assertion with his own evidence.

407. On 24th August 2011 Mr West saw his GP, complaining of eye symptoms. On examination, the GP noted the complaint of soreness, but recorded that *"there was not much to see"*. Mr West claimed that he had a long conversation with his GP about his other symptoms, about possible causes for them, including the fire, and that he was self-medicating. If that was the case, the GP would surely have made some note of it.

408. In my judgment, Mr West was a poor witness who was neither credible nor reliable. His evidence was replete with exaggeration and, at times, evasion. The weaknesses in his oral evidence will need to be balanced against the plume modelling evidence.

Mrs Kathleen Tully

409. Mrs Tully was born on 12th January 1970 and lives 1.53km from the Sonae plant. She has a history of coughs, respiratory tract infections, chest pain (but not for some years), conjunctivitis and sore throat.

410. Mrs Tully said that she suffered from a number of symptoms following the fire, including a sore throat, breathing problems, a chesty cough, sore eyes and a congested nose.

411. In her oral evidence, Mrs Tully described a very intense, acid-y smell and smoggy, heavy conditions. It looked like the day after bonfire day, and cars were white with dust.

412. On 16th June 2011 Mrs Tully went to see her GP. His computerised records reads as follows:

> "**Diagnosis** Ganglion of wrist right-sided, noticed a few weeks ago, some constant discomfort, able to use hand normally, no pins/needles. O/E – ganglion, radial – N, sensation – N. Explained and reassured.
>
> **Symptom** Has a sore throat last 3 days, no fever, feels achy all over, able to eat and drink a little, some diarrhoea, foul smelling, no blood. O/E -well hydrated, systemically well, throat – tonsils inflamed, pus bilate[ral] explained bacterial tonsillitis …"

413. Under cross-examination, Mrs Tully said that she had made a connection between her symptoms and the fire, and when she put this to her GP he said that he was not sure. Mrs Tully said that she was sure it was then, but immediately retracted this and said that it was possible that she mistaken about it. Given that there is no mention of this account in her witness statement, I have difficulty in accepting it.

414. On 27th June Mrs Tully returned to her GP complaining of the symptoms of acute conjunctivitis *"for 5/7 now"*. She had sore, itchy eyes. As regards the timing, Mrs Tully said in evidence that she did not think that her eye symptoms started on 22nd June, and that she must have misremembered the position when she spoke to her GP. She did associate her eye symptoms with the fire, but was unsure why she did not mention this to the doctor on 16th June.

415. In Dr Hardy and Dr Hind's opinion, Mrs Tully suffered from severe bacterial tonsillitis which was unrelated to the fire. Mr Swift's contrary conclusion that there was a causal relationship with smoke inhalation was based only on clinical judgment and the temporal association. He said that the GP must have failed to investigate the matter with sufficient care. Given the signs on examination, the clear presentation of a severe bacterial infection, and his own expert judgment that such infections are not the result of smoke inhalation, in my view Mr Swift's approach rather demonstrates the dangers of relying on *ex post facto* clinical judgments in this sort of situation. Nor can I accept Mr Clearkin's diagnosis of tear-film instability. The clinical findings, Mr Marsh's sound opinion, and Occam's Razor all point strongly in favour of an unrelated bacterial conjunctivitis.

416. Mrs Tully was a pleasant and entirely reasonable witness. I will be examining the plume modelling evidence in her case, but all the reliable and compelling evidence in her case points towards this being a coincidental infection.

Master Bradley Woods

417. Bradley was born on 30th April 2005 and at the time of the fire was aged only 6. He lives with his mother and sister 0.8km from the Sonae plant.

418. Bradley is reliant on the witness statement of his mother and Litigation Friend, Mrs Kathleen Woods. According to his questionnaire, Bradley developed respiratory, eye and skin problems within 24 hours of the fire. He had a persistent cough and complained of a frequent wheeze. His eyes became very itchy following the fire, and he developed a temperature. Bradley's symptoms had resolved within 2 weeks of the fire.

419. Mrs Woods does not have other than a vague recollection of conditions in the days following the fire, although she spoke of light grey smoke and "ashy" conditions outdoors.

420. On 14th June 2011 Bradley was taken to see his GP, who recorded that he had been coughing and suffering from a temperature for two days. This is inconsistent with the questionnaire. The GP diagnosed an upper respiratory tract infection.

421. Mrs Woods agreed that she did not mention her son's eyes to the GP: her explanation was that she was just bathing them.

422. Bradley clearly did suffer from a coincidental viral infection which was properly diagnosed on 14th June. It is of course possible that smoke inhalation might have aggravated his symptoms from this, but I would only be prepared to reach such a conclusion on the back of, at the very least, compelling plume modelling evidence supporting an above threshold exposure.

Mr Steven Woolvine

423. Mr Woolvine was born on 2nd June 1984. He lives 2.59km from the Sonae plant, and works as a customer service advisor at a location 1.65km from the fire.

424. Mr Woolvine has a history of hay fever, childhood eczema and dry skin complaints. He is a moderate smoker.

425. Mr Woolvine's solicitors throughout have been Walter Barr. There are two questionnaires relevant to his claim. Walter Barr told Mr Woolvine that this was due to *"administrative error"*. The first questionnaire, dated 8th August 2013, was completed and signed by Mr Woolvine. The second questionnaire, dated 16th September 2013, was neither signed nor completed by him. It contains several errors, and Mr Woolvine told me on oath that the signature is not his. I accept his evidence. It follows that Walter Barr, or their agents, have forged Mr Woolvine's signature. This is a serious matter, and I direct that a copy of his judgment be sent to the Solicitors Regulation Authority for investigation of this issue.

426. According to the first questionnaire, Mr Woolvine developed symptoms *"right away"* and these comprised cold sores, a cough, chapped lips, a chest infection and an eczematous rash. He claimed to have received medical treatment at his GP surgery. This questionnaire is vague and unsatisfactory.

427. Mr Woolvine's witness statement does provide further detail. He said that he developed a range of breathing, eye and skin problems in consequence of the fire. They were particularly bad for the first couple of weeks, and took approximately two months to resolve.

428. Under cross-examination, Mr Woolvine accepted that he did not make the connection between the fire and his symptoms until April 2013. He said that his eye and chest symptoms resolved within two weeks, but it took two months for the skin problems to recover.

429. Mr Woolvine saw his GP on 8th July 2011 in relation to an unrelated matter. He accepted that his questionnaire was incorrect in relation to medical treatment, and explained that he had misunderstood it.

430. Mr Woolvine was an unimpressive witness. Counsel began to fathom the bottom of his case during this sequence of cross-examination:

> "Q. Why didn't you go to the GP before 8 July, if you had the symptoms you've talked about?
>
> A. Because the symptoms that I had, I felt were manageable. I don't want to go to the GP about every little thing, because I obviously go quite a lot with regards to my depression and anxiety. I don't want to bombard him with every little thing.
>
> Q. No. So your cough never got so bad that you thought, well, I really need to go and see a doctor?
>
> A. No.
>
> Q. Nor your shortness of breath?

A. No. Because at that time my shortness of breath, initially I put it down to smoking, until it carried on for a couple of weeks, and then, because of the other symptoms I got, I related it to the fire.

Q. It's just it was suggested at one point in the papers [the account given by Mr Woolvine to Dr Hardy] that at one point you got so breathless you had to sit down on a wall when you got to the church because you were too breathless; is that right or not?

A. Yes. That's correct.

Q. If you really had that symptom at this time, you would have gone to the GP, wouldn't you?

A. No, because, as I say, initially, I did put it down to smoking and I did try and cut down at that stage, and after the two weeks it did ease off.

Q. So when you cut down the cigarettes, the symptoms reduced?

A. Yes, but I've never had problems -- I've smoked for 10, 12 years, on and off, so I've never experienced problems like that before, and it was just a bit coincidental that that happened at that period.

Q. The association you made between your symptoms and the fire came very much later, didn't it? The association in your mind that the fire may have caused problems came much later?

A. When I properly thought about it, yes."

431. In my judgment, Mr Woolvine grossly exaggerated his symptoms to Dr Hardy, and he has also given an exaggerated account to me. I entirely accept that this conclusion does not rule out the possibility of less severe symptoms resulting from exposure to the smoke plume, which symptoms may have crossed the threshold of actionable injury. However, as with other Test Claimants in his position, Mr Woolvine's difficulty is that he has to invoke something other than his oral evidence to persuade me of that proposition.

432. Having reviewed the evidence of the Test Claimants, and reached provisional conclusions in relation to many of them, this is now the appropriate moment at which to return to the plume modelling evidence.

THE REMODELLING BY THE PLUME MODELLERS: RESULTS AND DISCUSSION

433. On 7th July 2015 the results of CERC's plume remodelling exercise became available, and I invited further written submissions from the parties to be provided within seven days.

434. It is clear from Appendix 2 that CERC has followed my directions to the letter. I have included, unedited, their explanations of their work, for ease of comprehension. CERC's results may be summarised as follows.

435. Looking first of all at acrolein, it is clear that the majority of the Test Claimants were exposed to levels greater than the odour recognition threshold for many hours, the maximum being 44 hours (for Shaun West). The minimum levels of hourly exceedances are for Jessica Alexander (6 hours), Kelly Colebourne (2 hours), Karen Court (3 hours), Terence Dunn (4 hours), Teri O'Brien (3 hours), James Reece (zero) and Steven Woolvine (2 hours). However, as I have already said the odour recognition threshold is not a health threshold; for that, and as an indicative starting-point, I have taken a value of 146 μg/m³ for acrolein. The maximum theoretical hourly concentration for any Test Claimant is 12.01μg/m³, i.e. more than 10 times below this level. In fact, this Test Claimant is Peter Shaw who happened not to be at work on that day, and so was not exposed to such a dose. The second highest concentration is 9.48 μg/m³ (for Shaun West); for the vast majority of the Test Claimants the exposures are below 3 μg/m³. I appreciate that the 146 μg/m³ threshold is a trigger value, and not an hourly mean, but given the nature of this fire I doubt whether over relatively short periods of time this makes a significant difference. It would only have done so if the wind had changed over the course of any relevant hour.

436. A consideration of the 8-hour and 15-minute threshold values for acrolein of 237.5 μg/m³ and 712 μg/m³ respectively (see paragraph 142 above) demonstrates that all the Claimants come nowhere near these figures.

437. The position is very similar as regards the total aldehydes. In this instance, there are no examples of hourly exposures above the 500 μg/m³ threshold; indeed, the maxima are 41.0 (for Gary Mangan), 63.2 (for Peter Shaw – not that he was there), and 49.9 (for Shaun West).

438. As regards the PM_{10}s, the position is less unfavourable from the Claimants' perspective, but still not propitious. Here, I have taken the COMEAP trigger value of 107 μg/m³. In their remodelling, CERC has included the Briery Hey background concentrations because this was in accordance with my directions and ordinary tortious principles. For the vast majority of the Test Claimants, their maximum exposures were on 17th June 2011, and their highest recorded level was 100 μg/m³ (for a single hour – as to which, see paragraph 443 below). The modelling for these Claimants does not show two consecutive hours' exceedances; indeed, in most instances the hourly exposures were in the range of 10-50 μg/m³ (and typically within the band of 20-30 μg/m³) - in other words, at or scarcely above the background concentrations recorded at Briery Hey. There are a limited number of Test Claimants who fare somewhat better (in the forensic sense) with regard to the PM_{10}s. On 12th June, Gary Mangan incurred three consecutive hours' exposures of 103.6, 114.8 and 101.9 μg/m³ respectively. However, COMEAP would not have placed him at risk inasmuch as he was not exposed to two consecutive hours above 107 μg/m³. On 10th June, Peter Shaw (had he been at work) would have been exposed to a maximum concentration of 140.1 μg/m³. He would not been exposed to two consecutive hours

over 107 μg/m³. On 12th June, Shaun West was exposed to two sets of three consecutive hourly concentrations which were very close to, or above, the 107 μg/m³ level. On 17th June he was exposed to one single hour's concentration of 100.9 (although on that occasion the Briery Hey background level was high – 100.0).

439. As regards the $PM_{2.5}$s, the position is broadly similar. Strictly speaking, Gary Mangan cannot demonstrate exposures to levels above the COMEAP trigger value of 74 μg/m³ (for two consecutive hours), but he comes extremely close. Shaun West was above the trigger value on two occasions on 12th June. No one else threatens these levels.

440. CERC has also calculated the 24-hour mean concentrations for the five most heavily exposed Test Claimants. For Messrs Shaw and Swift, and Master Bradley Woods, their exposures were well below the CERC guideline values for the PM_{10}s (of 51 μg/m³ for vulnerable persons and 76 μg/m³ for non-vulnerable) and for the $PM_{2.5}$s (of 36 μg/m³ for vulnerable persons and 55 μg/m³ for non-vulnerable). Moreover, their exposures were not significantly above the Briery Hey background levels. However, on 12th June Gary Mangan's 24-hour mean concentrations were 54.3 μg/m³ for the PM_{10}s and 35.8 μg/m³ for the $PM_{2.5}$s. Likewise, on the same day Shaun West's mean concentrations were 65.6 μg/m³ and 45.0 μg/m³ respectively. Thus, it is open to them argue that there were certainly in the vicinity or "ball-park" of the 24-hour thresholds, depending on whether they should be regarded as vulnerable.

441. It is no accident that Gary Mangan and Shaun West were the most heavily exposed: they live relatively close to the Sonae plant. Interestingly, the comparable data for Dawn Bunting - as the crow flies, being the closest of all the Test Claimants to the source of the emissions - demonstrate a materially different pattern. Save for 17th June, her exposures to the PM_{10}s were broadly similar to the bulk of the Test Claimants', namely not substantially above background concentrations. The fact that her home is approximately 200m south of Messrs Mangan and West materially affects her exposure levels. When one then examines the positions of Paul McLoughlin, Bradley Woods and Kathleen Tully, being the sub-group of Test Claimants next closest to the plant, their exposures to the PM_{10}s and the $PM_{2.5}$s were scarcely distinguishable from those of the remainder, and much lower than Messrs Mangan's and West's. This exercise shows that only a very small coterie of Claimants living north-west of the plant were close to being within the "scientific" envelope of risk, and then only for relatively brief periods on 12th June (I exclude from account 17th June in relation to Shaun West, owing to the high background levels).

442. The contour maps within Appendix 2 illustrate the same point in a different way. In relation to the micro-particles, these maps show that anyone living further away from the plant than Gary Mangan received no exposures higher than the COMEAP trigger values. On my reckoning, a maximum of 250 individuals could have been living within the outer contour line. Their cases have not been individually examined, but it is possible that some of these could demonstrate two consecutive hours' exposure above the COMEAP trigger values. That, without more, would not be sufficient to prove actionable injury, but it would at least be a modest platform. Put slightly differently, such individuals *might* have the makings of a claim, but success would depend on their personal circumstances. I return to this issue at paragraph 465 below.

443. In his closing arguments delivered upon the conclusion of the oral evidence, Mr Kent submitted that the Briery Hey monitoring station did not show a "spike" in PM_{10}s in

consequence of the fire. Mr Redfern did not contradict that submission the following day, and it is borne out by the remodelling data. For example, as regards those Test Claimants who live nearest to Briery Hey, namely Jessica Alexander, James Glascott and Julie Carney, there is no evidence of any "spike" once the known Briery Hey values are removed from account. Further, I agree with Mr Kent that the 100 μg/m³ maximum value consistently referable by the remodelling to 17th June must be attributed to background levels rather than to the fire, and that the same analysis applies to the $PM_{2.5}$s on 5th July, where the remodelled value is about 66 μg/m³.

FINAL ANALYSIS, SYNTHESIS AND CONCLUSIONS

444. Viewed in isolation, the scientific evidence in this case does not begin to support these claims, save possibly for those of Gary Mangan and Shaun West. Indeed, viewed in those terms this evidence demonstrates that virtually all the Test Claimants could not have suffered actionable injury.

445. The key question which arises is whether I should be lowering the exposure thresholds to reflect scientific uncertainty, the difference between the scientific and the legal standards of proof, the quality of the Test Claimants' lay evidence and/or the views of Dr Hardy, Mr Swift and Mr Clearkin.

446. The matters I have identified as being potentially to the Claimants' advantage need to be addressed both individually and cumulatively.

447. Plainly, we are not in the realm of what might be called hard-edged science. An element of uncertainty has afflicted the exercise at every stage: as to Dr Mitcheson's heat release rates; the selection of the appropriate emission factors; the toxicology (at these low-exposure levels); and the plume modelling. However, three points need to be made. First, in relation to these issues I have applied a liberal approach throughout this judgment: that is to say, an approach which favours the Claimants where the evidence permits it. Secondly, the vast majority of the Test Claimants have fallen a long way short of demonstrating any significant exposures. Thirdly, there is no obvious reason why the Claimants should be the greater victims of scientific uncertainty than the Defendant. In my view, uncertainty is likely to be normally distributed.

448. I have mentioned the scientific uncertainties in connection with the ascertainment of the toxicological thresholds for low exposures. A related question arises as to whether the thresholds should be lowered to reflect pre-existing vulnerability. Many of the Test Claimants did not enjoy good general health, despite some of their assertions to the contrary. Many may have been prone to respiratory problems, and all those who were seen by the ophthalmologists have current signs of meibomian gland dysfunction. A number of Test Claimants, in particular Edmund Kenny, Kelly Colebourne and Annette Farrell, had pre-existing asthma. However, putting that aside for one moment, there is a lack of any solid, quantitative basis for making a discount for vulnerability. Many Claimants might have been vulnerable, but there is no proper means of assessing or quantifying this. Dr Hardy asserted that those without pre-existing pathologies tended to recover within 3-6 weeks of exposure, whereas those who were vulnerable required 3-6 months; but that assertion is simply not borne out

by a close examination of the circumstances of their individual cases, or indeed a proper identification of how and why they were "vulnerable". In any event, the level of discount must be an exercise in speculation, and for the vast majority of Test Claimants it would have to be very considerable for them to get anywhere. I note too that the differences between the COMEAP thresholds for vulnerable and non-vulnerable individuals are not great, and certainly well short of the magnitudes which would be required to avail any vulnerable Test Claimant.

449. A related point is that the available evidence fails to demonstrate any dose-related response. Listening as I did to all the Test Claimants give their evidence, the accounts they gave of the smoke plume and of the nature and severity of their symptoms bore no relationship with their modelled exposures. The smell and duration of the smoke was described in more or less exactly the same way irrespective of location. Indeed, I might be forgiven for thinking that some of the Test Claimants who in fact lived the furthest away from the plant were almost adjacent to it. Overall, the claimed symptoms appeared to be independent of exposure and dose. It was as if the Test Claimants had experienced a monolithic phenomenon. I appreciate that human beings differ, but I would expected to have heard accounts which varied according to the now ascertained doses.

450. Secondly, I do not consider that the application of a lower standard of proof requires me to approach the scientific evidence in any different way, save to recognise the scale of the potential uncertainties. I have already addressed that issue in paragraph 447 above. Where the law differs from the science is the weight it chooses to accord to lay evidence. This brings me to the third point.

451. In my judgment, the lay evidence viewed as a whole was unimpressive. It was vague, impressionistic, imprecise, sometimes inconsistent with the known behaviour of the smoke plume, and often internally inconsistent. Only three of the Test Claimants gave evidence which impressed me as being potentially reliable. Given that no one appears to have kept a contemporaneous record of his or her experiences, this generic failure to provide a coherent, consistent account of what occurred is hardly surprising, but in my judgment cannot be a factor in the Claimants' favour.

452. Had there being a critical mass of impressive, reliable lay evidence from the Claimants, I might have been prepared to revisit the toxicological thresholds and the plume modelling evidence. In the absence of these *desiderata*, I have absolutely no proper basis for lowering the bar.

453. The three Test Claimants who did impress me were Messrs Dunn, Kenny and McLoughlin. They all live north-west of the Sonae plant, but some considerable distance away. Mr Dunn, in many ways the most impressive witness, lives 2.3km away. His exposures were modest. Mr McLoughin gave quite convincing evidence in relation to his skin problems, but in the end I cannot accept that the science could be so wrong that he could be right. Mr Redfern submitted that the absence of any relationship between the good cases/witnesses and the modelled exposures somehow avails the Claimants, but in my judgment the true position is exactly the converse.

454. This brings me to the "constellation of symptoms" effect, and Dr Hardy's eloquent foray into quasi-epidemiology (Mr Swift and Mr Clearkin were less impassioned, but gave evidence to like effect). The difficulty with these arguments is that they entail an

overly generous and macroscopic view of the Test Claimants' evidence. Ultimately, they are founded on the apparent temporal association. What I have to call this ersatz epidemiology falls away as soon as the individual cases are scrutinised with the care they deserve in a forensic setting. As soon as that process occurred, the inconsistencies and weaknesses in most of the Test cases became evident. Furthermore, as soon as the plume modelling evidence is factored into the equation – as it has never been by the Claimants' clinical team – the claims become weaker still. In my judgment, these experts have sought to discern a constellation by gazing hopefully into the sky, without taking time to look closely at the individual stars.

455. The possibility arises that the science is just plain wrong, and that in many years' time a proper epidemiological study will prove that the residents of Kirkby were right all along. I cannot exclude that possibility. However, I have to use the best scientific evidence that is available, and then balance it against the lay evidence. Performing that exercise, I am satisfied on the balance of probabilities that the lay evidence I have heard cannot outweigh the combined effect of the science. Indeed, I would put the matter higher than that – in my view, the lay evidence creates no significant dent into the science, the latter emerging unscathed.

456. In my judgment, there are serious weaknesses in the Claimants' overall case which I need to make explicit. I have already alluded to some of them. First, the case is severely damaged by the delay in bringing these claims and the absence of any contemporaneous evidence. Had 16,000 people really suffered symptoms of the severity claimed, one would surely have seen evidence of complaints to newspapers and to the local council, increases in GP attendances, and some contemporary record of a problem. None has been brought to my attention. Secondly, recall bias is always an issue in scientific research based on retrospective evidence, and this phenomenon is hugely magnified when one brings into the equation the obvious corollaries of the medico-legal component. Human beings are naturally susceptible and suggestible, particularly if they are made to believe that they form part of a coherent group with shared experiences, and if they risk none of their own resources in bringing a claim. The standard-form questionnaires asked a series of leading questions. Many of the questionnaires examined in the context of the Test Claimants were shown to be inaccurate and exaggerated, calling into question the objectivity and integrity of the whole process. Nor does the whole set up of pop-up shops and cold-calling of potential Claimants inspire any degree of confidence.

457. My concerns in this regard are heightened by the fact that two of the questionnaires were shown to bear forged signatures, and that whole families have been signed up, apparently willy-nilly, to the group. The Defendant has drawn my attention, through the evidence of Ms Adele Wilson, to the sort of behaviour that has been going on. According to paragraph 6 of her witness statement:

> "In approximately January 2012, I was at work when I received a telephone call from my partner, Greg Taft. Greg told me that a lady had visited our home in Tower Hill who had told him that she was acting on behalf of GT Law solicitors who was dealing with claims against Sonae relating to smoke inhalation from the fire in June 2011. She said that Sonae had accepted liability and that compensation had already been paid out to claimants. The lady was attempting to encourage Greg to sign

> up in order to put forward a claim against Sonae for symptoms relating to smoke inhalation. Greg told her that he was not interested in making a claim and asked her to leave."

Ms Wilson was not cross-examined about this evidence, which is admittedly hearsay. However, there is no reason to doubt its accuracy. The information Mr Taft was given was inaccurate – there had been no admission of liability, and no money had been paid. Misleading information of this sort had the obvious tendency to encourage the bringing of claims, on the basis that the Defendant was a soft target and this was easy money. That this information was understood in exactly this way is revealed by the terms of the Facebook posts referred to at paragraphs 9, 10 and 12 of Ms Wilson's witness statement, as well as by the evidence in Leon Swift's case. I strongly deprecate this sort of practice. Not merely does it sail close to the wind in terms of its professional propriety, it is severely counter-productive as and when the case comes to trial.

458. I have considered the possibility of an alternative narrative pursuant to which a number of Claimants might succeed. It runs like this. The GP records are all incorrect/inaccurate; the Claimants have short-changed themselves in terms of their timings and the exact sequence of events (they must not be criticised for not remembering); when all the inconsistencies, exaggerations and excrescences are stripped away, there lies a hard kernel of truth – a truth which says that many of the Claimants did suffer actionable injury, albeit maybe not for as long as they have claimed. In many cases, so this argument might run, Claimants did sustain exacerbations of pre-existing conditions.

459. Mr Redfern did not advance submissions along precisely these lines, for obvious reasons – he could not be heard to do so. However, it is clearly right that I should be considering this alternative viewpoint. Apart from the scientific evidence, which strongly tells against such a narrative being correct, there is this added difficulty. At its highest, the narrative I am postulating *might* just be true, but there is no basis for holding that it is *probably* true. Furthermore, there is no sensible or remotely plausible basis for concluding that so much hard evidence, including the contemporaneous GP record, is incorrect, and it simply would not be right to make findings of fact based on a version of events that does not match the evidence the Claimants chose to give.

460. I return to the cases of the three Test Claimants whose evidence was, as I have said, *prima facie* reliable. Given that the scientific evidence is so heavily against them, there is in my judgment no proper, principled basis for allowing their claims. That they were good witnesses is insufficient: that does not preclude their being mistaken (in my judgment, they *are* mistaken) and/or being sucked into the vortex of suggestibility created by the claims environment which obtained in Kirkby in 2013.

461. The strongest case out of the 20 is Mr Edmund Kenny's. He was an impressive witness and also immensely likeable. He did not exaggerate his symptoms in any way. It is clear that he did suffer a flare-up in his asthma symptoms in June 2011, and that he told his GP that he suspected a causal link. He accepted in cross-examination that he did not know whether his suspicions were correct. Ultimately, it is not his view which counts but the opinion of an expert in respiratory diseases. Plainly, neither Dr Hardy nor Dr Hind made a contemporaneous examination. Mr Kenny has the benefit of no contemporaneous medical examination, and retrospective diagnosis is

problematic. His exposures were low: scarcely above the background levels for the micro-particles (I have deduced that the majority of the "spike" on 17th June, and certainly the single 100 μg/m³ reading, is unrelated to the fire), and a maximum hourly concentration of 1.45 μg/m³ for acrolein. In my judgment, he fails to satisfy me of a causal link.

462. As for Gary Mangan and Shaun West, on account of their exposures they stood the best chance of proving actionable injury in consequence of the Sonae fire, but in my judgment their claims fail nonetheless. Mr Mangan is a fit young man who could not convincingly be described as vulnerable. The plume modelling measures out-of-door exposures, and Mr Mangan did not tell me that he ventured outside at the relevant time. His case lies at the margins of the PM_{10} trigger levels, but is considerably lower than the threshold levels for the irritants. In any event, the better view on the balance of probabilities is that he suffered from a coincidental upper respiratory tract infection. Mr West was not a good witness, and there were clear inconsistencies and exaggerations in his account. He too could not be described as vulnerable. The generic considerations set out at paragraph 465 below also apply.

463. Where does the truth lie in all of this? It is not my function to say what the truth is; my role is limited to deciding whether these claims succeed or fail, applying standard legal methods. However, out of fairness to the Claimants my conclusions about what happened in June 2011 are as follows. There is no unified field theory or overarching explanation. The majority of Claimants experienced an extremely nasty, unusual smell and some may have been concerned about the possible consequences, albeit not so concerned to seek medical advice. Many Claimants - it is not clear to me how many – suffered some symptoms of shortness of breath, lacrimation and soreness of the throat. These symptoms were short-lived, as were any significant quantities of smoke. For the majority of the time, the smoke was blowing away from the plant. The better view on all the available evidence is that these symptoms did not exceed the hurdle the law sets for actionable personal injury, because they were symptoms of irritation rather than of inflammation. It is difficult to say for how long the smoke and these mild symptoms lasted, but I have in mind a maximum period of about one week. Many months later – it is unclear exactly how and why - lawyers arrived on the scene and sensed the opening of a business opportunity. It proved not very difficult to recruit willing Claimants to the group, not least because there was a lot of ill-feeling in the neighbourhood directed towards Sonae, and many people genuinely believed that they must have been harmed in some way. The legal process preyed on human susceptibility and vulnerability, and the rest is history.

464. I accept that for a period of time, probably 2-3 days, there were deposits of ash and dust which proved recalcitrant and pervasive. It is regrettable that Dr Mitcheson was not cross-examined about this, but I am not prepared to disbelieve the entirety of the Claimants' evidence on this point. However, I should not be understood as excluding from account the probability of poetic licence and exaggeration.

465. Previously, I have referred to a maximum of 250 people living within the outer contour line on the CERC contour maps marking exposures to the micro-particles at above the COMEAP trigger values (albeit, I reiterate, not for two consecutive hours). I do not know how many of these are Claimants. I cannot exclude the possibility that some of these might have suffered personal injury, but I have three observations. First, the Claimants have only asked me to examine one individual within this sub-

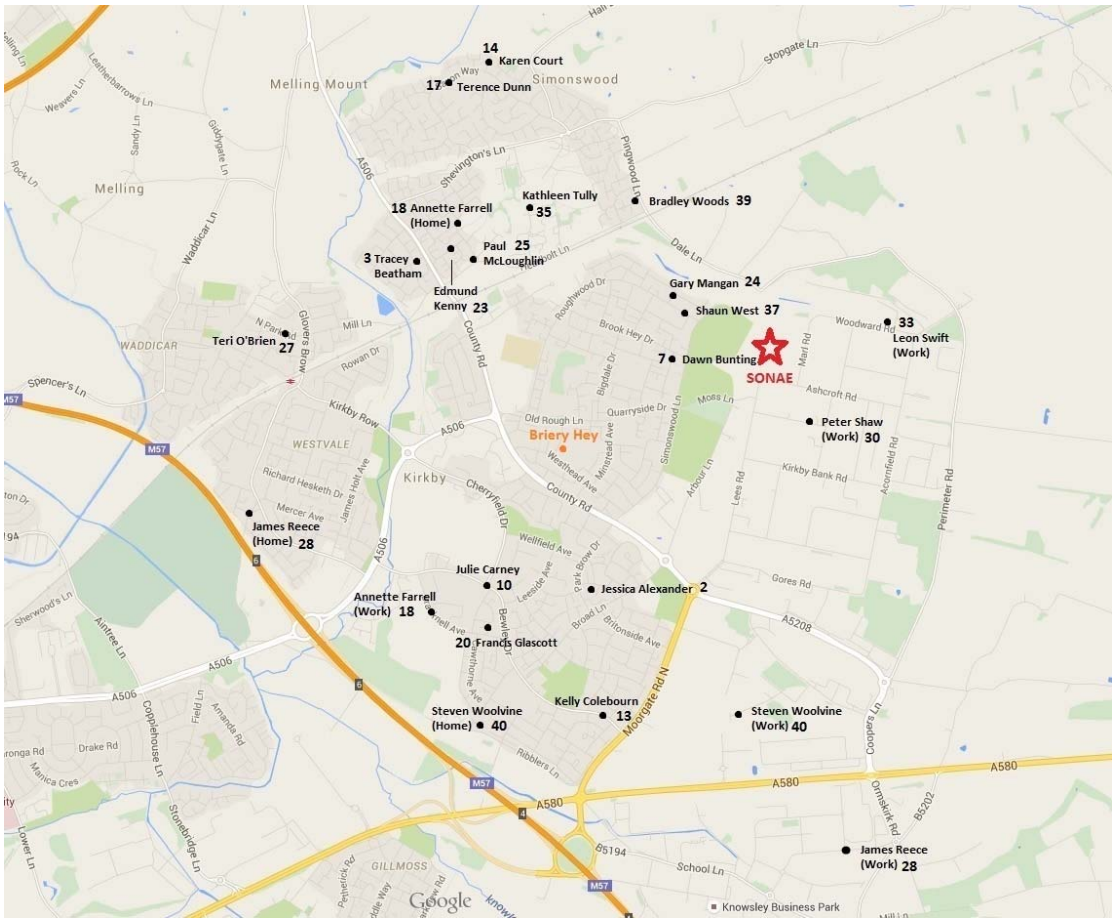
cohort, namely Gary Mangan. Presumably, he was selected because he was representative of the others, and/or because those advising the Claimants believed that he would be a good witness. My observation would be that if there were stronger cases within this sub-group, they should have been put forward as Test claims. Secondly, I suspect that only those with pre-existing conditions might have had a realistic prospect of proving actionable damage. I should not be interpreted as overlooking the fact that all these individuals fell vastly below the relevant health threshold for acrolein, and well below the PM_{10} levels at which significant numbers of children complained of symptoms as demonstrated in the Californian Wildfires study (see paragraph 157 above). Thirdly, after a multi-million pound group action which has failed, in my judgment it would scarcely be proportionate to start examining even a handful of these cases, in the pursuit of identifying what would be, at best, modest claims. In my judgment, the Claimants' chance has come and gone.

466. There are clear lessons to be learned from this litigation. The Claimants' legal team should have worked out the science at a much earlier stage. Working on a reasonable best case scenario (as I have done), sound toxicological evidence (which they had at their disposal) and plume modelling, they should have investigated whether the case stacked up. Instead, at all material times, the Claimants' legal team appear to have wanted to make a virtue out of uncertainty – perhaps because they clung to the notion that the litigation would settle. Alternatively, they believed that the judge would not be that interested in the science, and/or they placed undue faith on the likely cogency of the lay evidence.

467. On all counts, they have been proven wrong. Further, rather than give the Claimants the opportunity to rely positively on uncertainty, I have taken proactive steps to enable further modelling to be undertaken, thereby creating as robust an evidential basis as human ingenuity may currently provide. The Claimants have lost no money, but their expectations, always unsustainable in my view, have not been fulfilled. I regret that their hopes were raised in the first place.

468. These claims must all be dismissed, and judgment must be entered for the Defendant.

APPENDIX 1


14 Karen Court
17 Terence Dunn
Melling Mount
Simonswood
Melling
WADDICAR
WESTVALE
Kirkby
Briery Hey
SONAE
18 Annette Farrell (Home)
Kathleen Tully 35
Bradley Woods 39
3 Tracey Beatham
Paul 25 McLoughlin
Edmund Kenny 23
Gary Mangan 24
Shaun West 37
33 Leon Swift (Work)
7 Dawn Bunting
Teri O'Brien 27
Peter Shaw (Work) 30
James Reece (Home) 28
Julie Carney 10
Jessica Alexander 2
Annette Farrell (Work) 18
20 Francis Glascott
Steven Woolvine (Home) 40
Kelly Colebourn 13
Steven Woolvine (Work) 40
James Reece (Work) 28
Knowsley Business Park
A506
A580
A5208
M57
B5194
B5202
Google

CLAIMANT NUMBER	CLAIMANT NAME	POSTCODE	DISTANCE FROM SONAE (KM)
2	JESSICA ALEXANDER	L32 8TX	1.56
2	JESSICA ALEXANDER COLLEGE	L33 8XF	1.42
3	TRACEY BEATHAM	L33 2DE	1.96
7	DAWN BUNTING	L33 6XB	0.44
10	JULIE CARNEY	L32 9PQ	1.94
13	KELLY COLEBOURN	L32 7PZ	2.16
14	KAREN COURT	L33 4DH	2.27
17	TERENCE DUNN	L33 4DP	2.31
18	ANNETTE FARRELL	L33 1UW	1.81
18	ANNETTE FARRELL WORK	L32 9PP	2.27
20	FRANCIS GLASCOTT	L32 9QD	2.1
23	EDMUND KENNY	L33 1YF	1.8
24	GARY MANGAN	L33 9XF	0.53
25	PAUL MCLOUGHLIN	L33 1WD	1.61
27	TERI O’BRIEN	L32 2AR	2.63
28	JAMES REECE	L32 4SP	3.01
28	JAMES REECE WORK	L32 9HN	2.87
30	PETER SHAW WORK	L33 7TJ	0.55
33	LEON SWIFT	L33 7UY	0.88
35	KATHLEEN TULLY	L33 1UG	1.53
37	SHAUN WEST	L33 9UJ	0.58
39	BRADLEY WOODS	L33 1RF	1.09
40	STEVEN WOOLVINE	L32 7RP	2.59
40	STEVEN WOOLVINE WORK	L33 7RX	1.65

APPENDIX 2

Sonae GLO: Further Modelling

Contents

Following the instructions of Mr Justice Jay further modelling by CERC is presented in the accompanying seven documents. As specified by Mr Justice Jay, the modelling is based on:

- 1550 tonnes of material consumed in fire;
- The heat release and burning rates specified by Dr Mitcheson, with 10% subtracted to account for radiative heat loss
- An emission factor of 114 mg/s/MW for acrolein for all stages of the fire, compared against an odour threshold of 0.38 µg/m³ and a health threshold of 146 µg/m³
- An emission of 600 mg/s/MW for total aldehydes for all stages of the fire, compared against a health threshold of 500 µg/m³
- PM_{10} emission factors of 27.5 g/kg for Stage 2 and 12.5 g/kg for Stages 1 and 3 of the fire, compared against the COMEAP High trigger value of 107 µg/m³. Assuming 0.8% contamination, as specified by the Mr Justice Jay, the PM_{10} emission factors used are 27.61 g/kg for Stage 2 and 12.73 g/kg for Stages 1 and 3
- $PM_{2.5}$ emission factors of 22.6 g/kg for Stage 2 and 10.25 g/kg for Stages 1 and 3 of the fire, compared against the COMEAP High trigger value of 74 µg/m³. Assuming 0.8% contamination, as specified, the $PM_{2.5}$ emission factors used are 22.64 g/kg for Stage 2 and 10.39 g/kg for Stages 1 and 3
- The agreed meteorological data, of Dr Wild and Mr Lynagh (Lynagh/Wild dataset). CERC and Envirobods agreed that in order to make full use of the Lynagh/Wild dataset, hourly inputs for the model should be derived by calculating hourly averages from the 10-minute data recorded every 15 minutes, as detailed in CERC's supplementary report dated 22nd September 2014. The Lynagh/Wild dataset only provides data until 1st July 2011, therefore to consider whole period of the fire meteorological data from the Met Office Crosby station were used from the 1st July 2011, up to and including the 7th July 2011
- Modelled PM_{10} and $PM_{2.5}$ concentrations include background concentrations using values measured at the Briery Hey monitoring site. This monitoring data includes a 43 hour gap in $PM_{2.5}$ monitoring between the 10th June 2011 and 12th June 2011. $PM_{2.5}$ concentrations for this period were estimated from the measured PM_{10} concentrations, assuming a $PM_{2.5}/PM_{10}$ ratio of 0.45. This ratio is the ratio of the mean concentrations over the period of the fire, where both $PM_{2.5}$ and PM_{10} concentrations are recorded.

Based on this model set-up, specified by Mr Justice Jay and outstanding elements agreed between the dispersion modelling experts, the following outputs are provided:

1. Document 1: Contour maps of the number of hours exceeding the odour threshold for acrolein and the COMEAP High trigger values for PM_{10} and $PM_{2.5}$. Contours for exceedences of the health thresholds for acrolein and total aldehydes could not be plotted because modelled concentrations are not sufficiently high.
2. Document 2: Tables of number of hours exceeding the thresholds of PM_{10}, $PM_{2.5}$, acrolein and total aldehydes at the locations of the 20 Test Claimants, along with modelled maximum hourly concentrations at these locations.
3. Document 3: Tables of modelled PM_{10} and $PM_{2.5}$ concentrations for particular days and Test Claimant locations, as specified by Mr Justice Jay.
4. Document 4: Time series histograms of hourly 'unit discharge' concentrations compared against the specified acrolein and total aldehydes thresholds for the locations of the 20 Test Claimants.
5. Document 4a: Time series histograms of hourly 'unit discharge' concentrations, plotted for a lower concentration range than used in Document 4, to show modelled concentrations more clearly and allow better comparison against the acrolein odour threshold.
6. Document 5: Time series histograms of hourly $PM_{2.5}$ concentrations for the locations of the 20 Test Claimants, compared with the COMEAP High trigger value of 74 µg/m³.
7. Document 6: Time series histograms of hourly PM_{10} concentrations for the locations of the 20 Test Claimants, compared with the COMEAP High trigger value of 107 µg/m³.

Sonae GLO: Further Modelling

1. Contour maps of number of hours exceeding threshold values

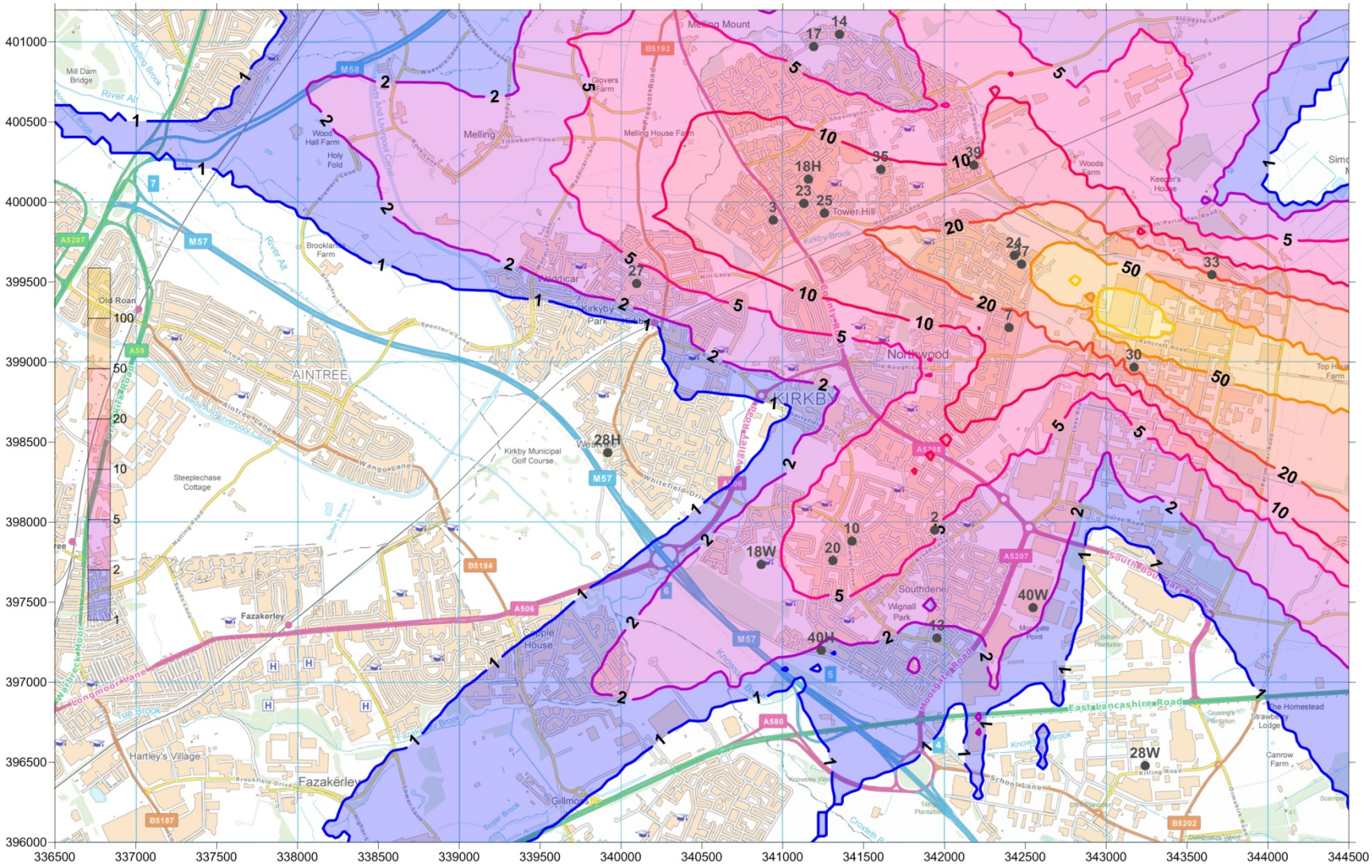
This document presents the following contour maps of modelled number of hours for which hourly average concentrations exceed specified threshold values:

- Acrolein concentrations exceeding the odour recognition threshold of 0.38 µg/m³
- PM_{10} concentrations, including background concentrations using measured data from the Briery Hey monitoring site, exceeding the COMEAP High trigger value of 107 µg/m³.
    - Two plots are presented for PM_{10}: the first is based on 100 m resolution model output for an output area of 9.4 km x 6.6 km; and the second is based on 50 m resolution model output for an output area of 5 km x 4 km, producing better resolution of the modelled exceedence area.
- $PM_{2.5}$ concentrations, including background concentrations using measured data from the Briery Hey monitoring site, exceeding the COMEAP High trigger value of 74 µg/m³.
    - Two plots are presented for $PM_{2.5}$: the first is based on 100 m resolution model output for an output area of 9.4 km x 6.6 km; and the second is based on 50 m resolution model output for an output area of 5 km x 4 km, producing better resolution of the modelled exceedence area.

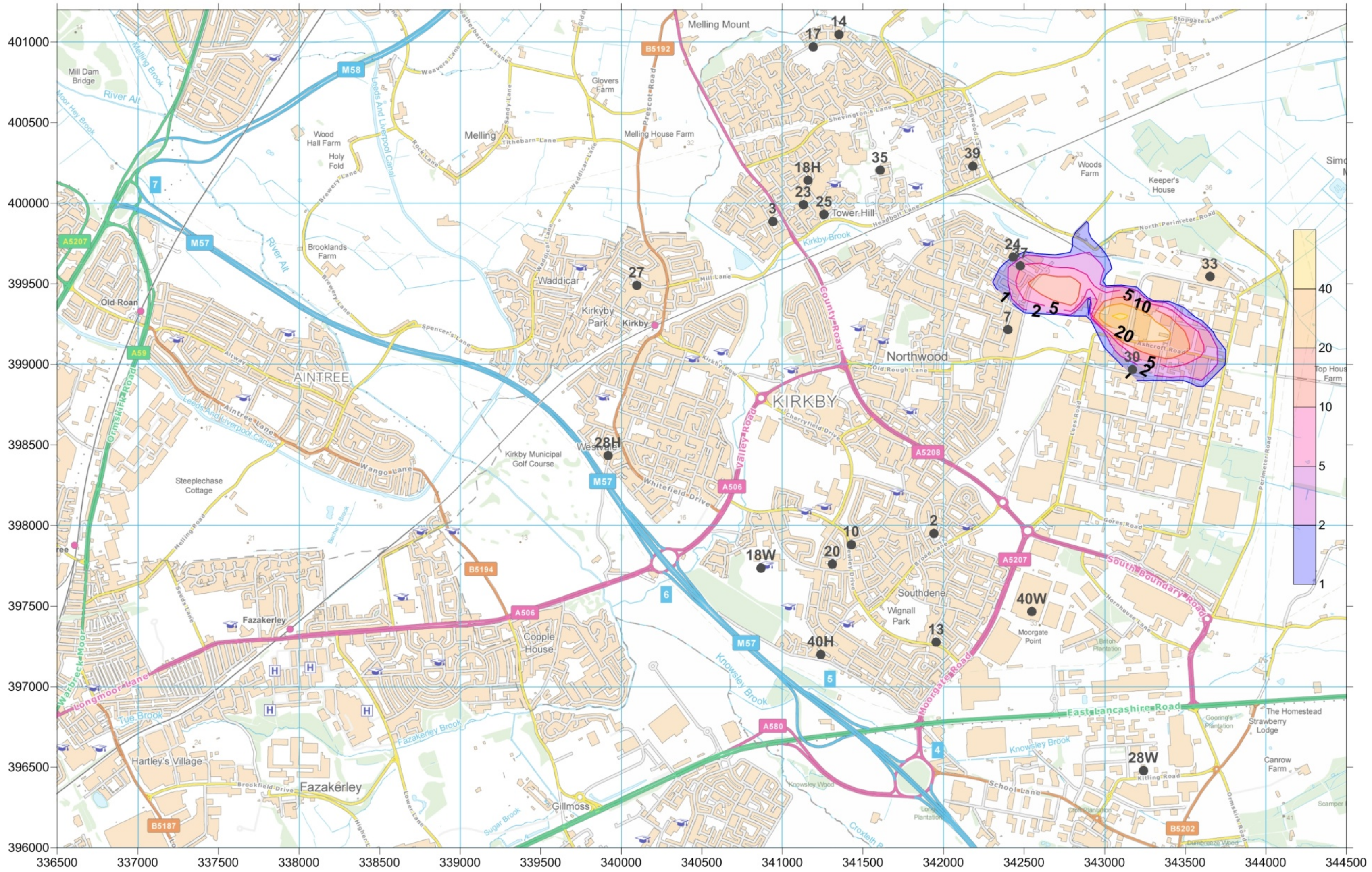
Contour maps for the acrolein health threshold of 146 µg/m³ and the total aldehydes threshold of 500 µg/m³ have not been produced because these thresholds are not exceeded beyond the boundary of the Sonae factory.
The maximum modelled acrolein concentration across the output area is 95.6 µg/m³, below the health threshold, therefore a contour map cannot be produced since there are no exceedences of the threshold across the model output area.[1]
The maximum modelled total aldehydes concentration across the output area is 502.6 µg/m³, just above the threshold of 500 µg/m³. This maximum concentration is modelled with the 50 m resolution output and is the only point across the output area above the threshold value. This output point is located within the boundary of the Sonae factory.
To minimise differences in the appearance of contour plots due to contouring methodology, CERC and Envirobods agreed to use Golden Software's Surfer package for contour plotting, using the Inverse Distance Weighting interpolation method.

[1] A contour map is produced interpolating changes in values in between the model output points. This calculation cannot be performed by the contouring software if all the output points are equal.

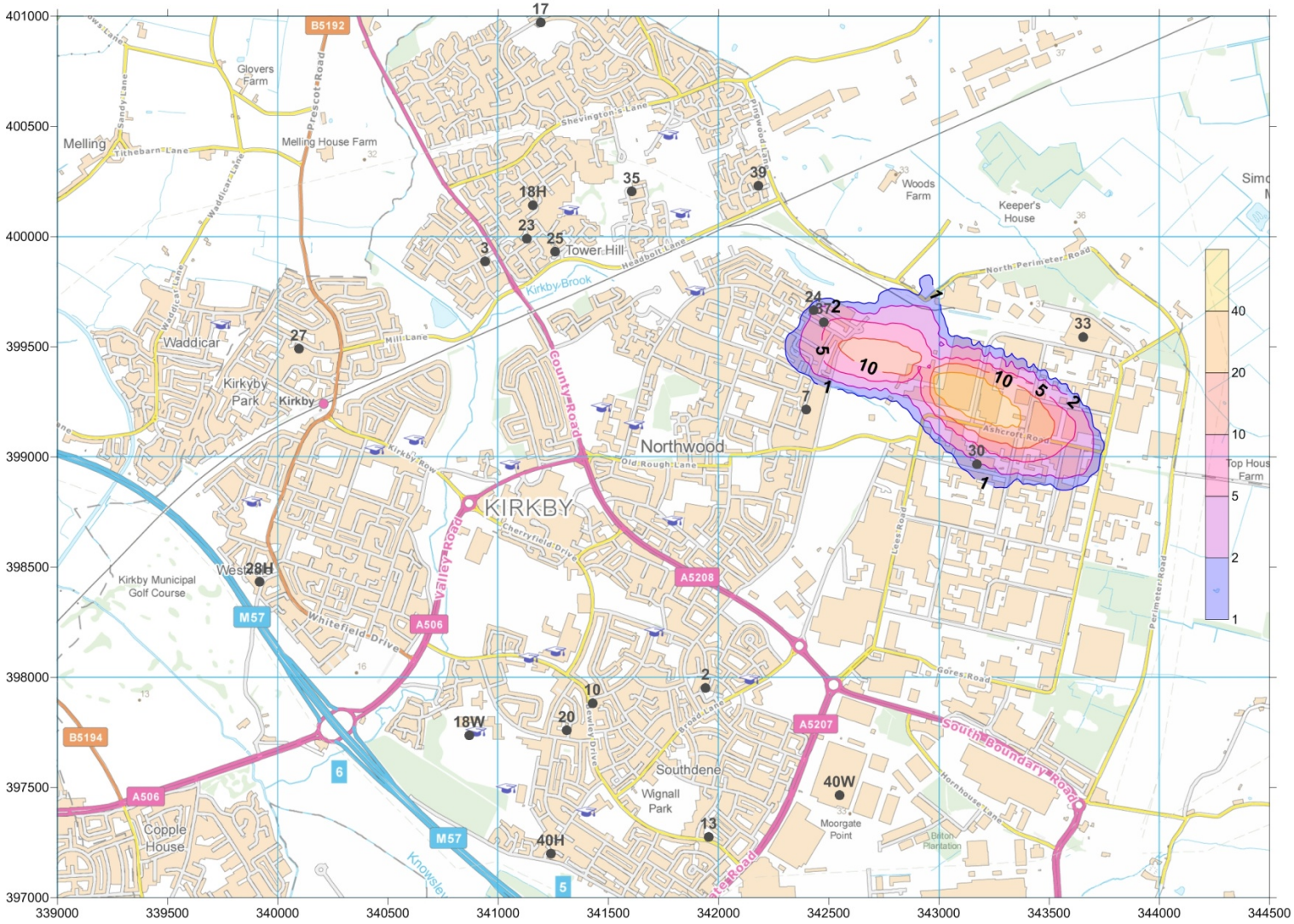
Number of hours for which the hourly average acrolein concentration exceeds 0.38 µg/m³ (odour recognition threshold)



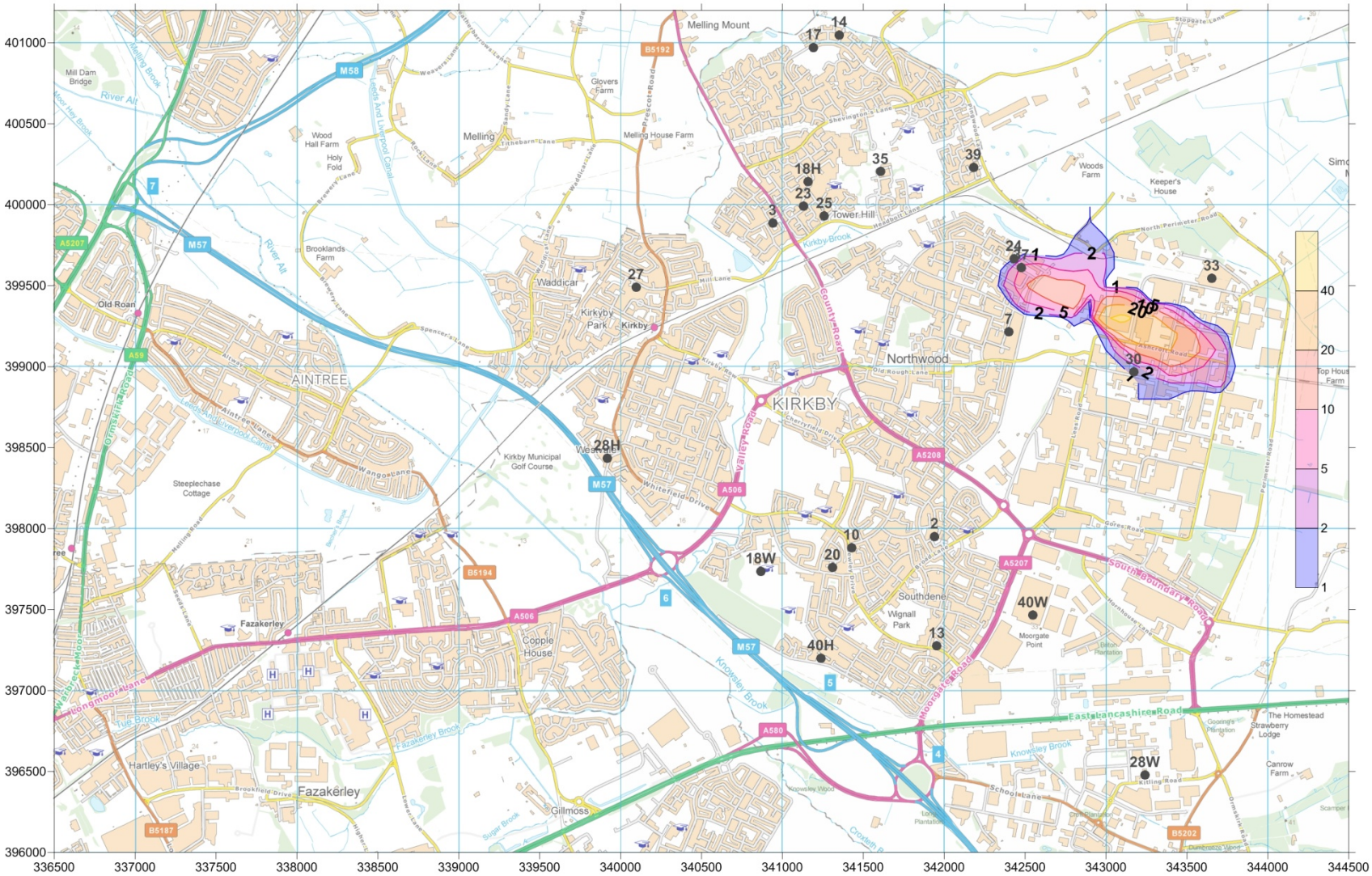
Number of hours for which the hourly average PM10 concentration exceeds 107 µg/m³ (COMEAP High trigger value); Modelled concentrations include background values from the Briey Hey monitoring site; 100 m resolution output



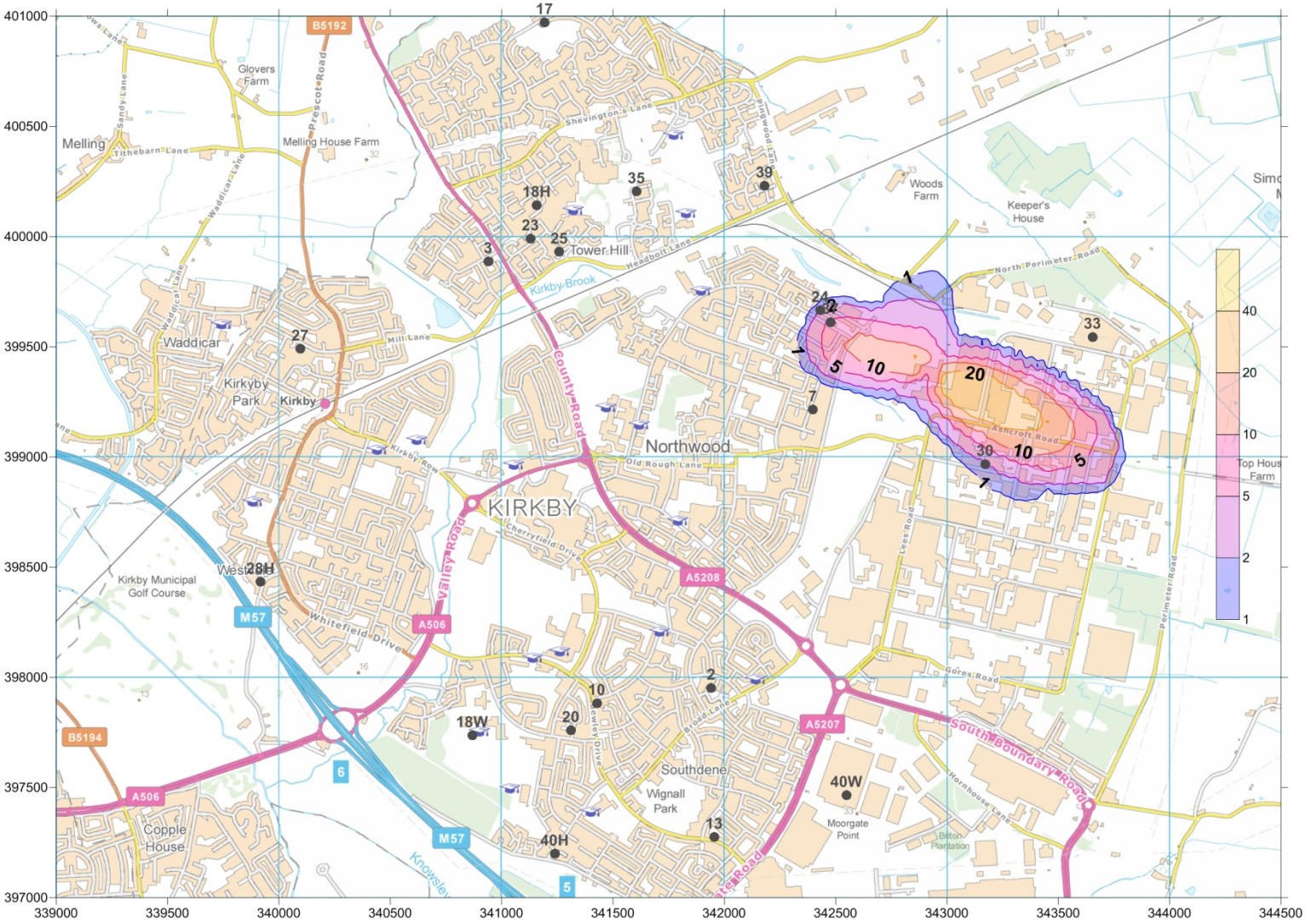
Number of hours for which the hourly average PM10 concentration exceeds 107 µg/m³ (COMEAP High trigger value); Modelled concentrations include background values from the Briey Hey monitoring site; 50 m resolution output



Number of hours for which the hourly average PM2.5 concentration exceeds 74 µg/m³ (COMEAP High trigger value); Modelled concentrations include background values from the Briey Hey monitoring site; 100 m resolution output



Number of hours for which the hourly average PM2.5 concentration exceeds 74 µg/m³ (COMEAP High trigger value); Modelled concentrations include background values from the Briey Hey monitoring site; 50 m resolution output



Sonae GLO: Further Modelling

2. Tables of modelled concentrations at the locations of the 20 Test Claimants

Table 1 lists the locations of 20 Test Claimants. Two tables then follow presenting the model outputs for the locations of the 20 Test Claimants:
Table 2 presents the number of hours exceeding the COMEAP High trigger values for PM_{10} and $PM_{2.5}$ and the maximum predicted hourly concentrations for these pollutants. The number of exceedences and maximum hourly concentrations include the modelled contribution from the fire and background concentrations using measured data from the Briery Hey monitoring site.
Table 3 presents the number of hours exceeding acrolein thresholds for odour (0.38 µg/m³) and health (146 µg/m³), and the health threshold for total aldehydes (500 µg/m³).

Table 1: Locations of the 20 Test Claimants

ID	Name	Postcode	Coordinates of location (Ordnance Survey British National Grid)	
			Eastings (m)	Northings (m)
2	Jessica Alexander	L32 8TX	341941	397952
3	Tracey Beatham	L33 2DE	340940	399888
7	Dawn Bunting	L33 6XB	342398	399215
10	Julie Ann Carney	L32 9PQ	341428	397883
13	Kelly Colebourn	L32 7PZ	341955	397276
14	Karen Court	L33 4DH	341351	401049
17	Terence Dunn	L33 4DP	341193	400972
18H	Annette Farrell (home)	L33 1UW	341158	400143
18W	Annette Farrell (workplace)	L32 9PP	340868	397737
20	Francis Glascott	L32 9QD	341311	397760
23	Edmund Kenny	L33 1YF	341130	399991
24	Gary Mangan	L33 9XF	342431	399666
25	Paul McLoughlin	L33 1WD	341259	399931
27	Teri O'Brien	L32 2AR	340096	399491
28H	James Reece (home)	L32 4SP	339918	398434
28W	James Reece (workplace)	L34 9HN	343241	396479
30	Peter Shaw	L33 7TJ	343172	398968
33	Leon Swift	L33 7UY	343654	399545
35	Kathleen Tully	L33 1UG	341606	400205
37	Shaun West	L33 9UJ	342477	399612
39	Bradley Woods	L33 1RF	342181	400231
40H	Steven Woolvine (home)	L32 7RP	341239	397200
40W	Steven Woolvine (workplace)	L33 7RX	342549	397466

Table 2: Number of hours exceeding COMEAP High trigger values for PM_{10} and $PM_{2.5}$, together with modelled maximum concentrations, at the test case locations. Modelled concentrations include background concentrations from the Briery Hey monitoring site.

ID	PM_{10} (including background concentration)		$PM_{2.5}$ (including background concentration)	
	Number of hours exceeding COMEAP High trigger value (107 µg/m³)	Maximum hourly concentration (µg/m³)	Number of hours exceeding COMEAP High trigger value (74 µg/m³)	Maximum hourly concentration (µg/m³)
2	0	100.0	0	66.0
3	0	100.0	0	66.0
7	0	100.0	0	66.0
10	0	100.0	0	66.0
13	0	100.0	0	66.0
14	0	100.5	0	66.0
17	0	100.3	0	66.0
18H	0	100.0	0	66.0
18W	0	100.0	0	66.0
20	0	100.0	0	66.0
23	0	100.0	0	66.0
24	2	114.8	2	79.9
25	0	100.0	0	66.0
27	0	100.0	0	66.0
28H	0	100.0	0	66.0
28W	0	100.0	0	66.0
30	1	140.1	1	112.2
33	0	100.0	0	66.0
35	0	100.1	0	66.0
37	5	135.8	6	96.4
39	0	101.0	0	66.0
40H	0	100.0	0	66.0
40W	0	100.0	0	66.0

Table 3: Number of hours exceeding odour and health thresholds for acrolein, and health threshold for total aldehydes at the test case locations. Modelled maximum concentrations for these pollutants are also presented.

ID	Acrolein			Total aldehydes	
	Number of hours exceeding threshold value		Maximum hourly concentration (µg/m³)	Number of hours exceeding health threshold (500 µg/m³)	Maximum hourly concentration (µg/m³)
	Odour (0.38 µg/m³)	Health (146 µg/m³)			
2	6	0	0.74	0	3.9
3	16	0	1.37	0	7.2
7	10	0	1.40	0	7.4
10	10	0	1.05	0	5.5
13	2	0	0.59	0	3.1
14	3	0	0.82	0	4.3
17	4	0	1.17	0	6.2
18H	14	0	1.29	0	6.8
18W	4	0	0.96	0	5.1
20	9	0	0.97	0	5.1
23	18	0	1.45	0	7.6
24	36	0	7.78	0	41.0
25	18	0	1.55	0	8.2
27	3	0	0.66	0	3.5
28H	0	0	0.25	0	1.3
28W	0	0	0.30	0	1.6
30	14	0	12.01	0	63.2
33	17	0	2.21	0	11.6
35	11	0	1.83	0	9.6
37	44	0	9.48	0	49.9
39	9	0	1.46	0	7.7
40H	2	0	0.39	0	2.1
40W	2	0	0.49	0	2.6

Sonae Group Litigation: Final Modelling

3. Tables of modelled hourly PM_{10} and $PM_{2.5}$ concentrations for specific days and Test Claimants

This document presents tables of PM_{10} and $PM_{2.5}$ concentrations for the day and Test Claimant combination specified in Table 1. Hourly concentrations and 24-hour mean concentrations are presented along with background concentrations from the Briery Hey monitoring site.

Table 4: Summary of daily PM_{10} and $PM_{2.5}$ concentrations tables presented in this document

Test Claimant ID	Day	Table number
24	10th June 2011	2
	12th June 2011	3
30	10th June 2011	4
	11th June 2011	5
	27th June 2011	6
33	11th June 2011	7
	15th June 2011	8
	20th June 2011	9
37	10th June 2011	10
	12th June 2011	11
	17th June 2011	12
39	10th June 2011	13
	12th June 2011	14
	17th June 2011	15

Table 5: Modelled hourly PM_{10} and $PM_{2.5}$ concentrations for Test Claimant 24, 10th June 2011

10/06/2011	Test Claimant 24			
Hour (hour ending BST)	Modelled concentrations including background (µg/m³)		Briery Hey background (µg/m³)	
	PM_{10}	$PM_{2.5}$	PM_{10}	$PM_{2.5}$
1	18.3	6.9	16.0	5.0
2	16.0	10.0	16.0	10.0
3	18.0	1.0	18.0	1.0
4	32.6	20.8	17.0	8.0
5	20.0	5.0	20.0	5.0
6	31.5	9.4	31.0	9.0
7	22.0	8.0	22.0	8.0
8	30.0	6.0	30.0	6.0
9	26.0	1.0	26.0	1.0
10	28.0	11.0	28.0	11.0
11	13.6	7.1	11.0	5.0
12	12.0	8.0	12.0	8.0
13	15.0	6.8	15.0	6.8
14	6.0	2.7	6.0	2.7
15	17.0	7.7	17.0	7.7
16	19.0	8.6	19.0	8.6
17	20.0	9.0	20.0	9.0
18	19.0	8.6	19.0	8.6
19	3.0	1.4	3.0	1.4
20	7.0	3.2	7.0	3.2
21	13.0	5.9	13.0	5.9
22	9.0	4.1	9.0	4.1
23	5.0	2.3	5.0	2.3
24	19.0	8.6	19.0	8.6
24-hour mean concentration (µg/m³)	**17.5**	**6.8**	***16.6***	***6.1***
24-hour mean concentration excluding background (µg/m³)	**0.9**	**0.7**		

Table 6: Modelled hourly PM_{10} and $PM_{2.5}$ concentrations for Test Claimant 24, 12th June 2011

12/06/2011	Test Claimant 24			
Hour (hour ending BST)	Modelled concentrations including background (µg/m³)		Briery Hey background (µg/m³)	
	PM_{10}	$PM_{2.5}$	PM_{10}	$PM_{2.5}$
1	16.0	7.2	16.0	7.2
2	21.0	9.5	21.0	9.5
3	20.0	9.0	20.0	9.0
4	18.0	8.1	18.0	8.1
5	21.0	9.5	21.0	9.5
6	20.0	9.0	20.0	9.0
7	32.9	16.9	27.0	12.2
8	64.4	28.2	36.0	5.0
9	42.9	17.9	32.0	9.0
10	103.6	68.4	26.0	5.0
11	114.8	79.2	30.0	10.0
12	101.9	71.9	15.0	1.0
13	49.2	42.2	11.0	11.0
14	68.1	47.8	23.0	11.0
15	32.0	20.4	18.0	9.0
16	35.3	28.4	14.0	11.0
17	22.1	6.0	16.0	1.0
18	75.3	46.8	18.0	0.0
19	82.7	63.1	14.0	7.0
20	87.9	65.4	20.0	10.0
21	95.5	71.3	18.0	8.0
22	109.2	79.9	26.0	12.0
23	59.0	45.1	16.0	10.0
24	10.4	7.3	10.0	7.0
24-hour mean concentration (µg/m³)	**54.3**	**35.8**	***20.2***	***8.0***
24-hour mean concentration excluding background (µg/m³)	**34.1**	**27.8**		

Table 7: Modelled hourly PM_{10} and $PM_{2.5}$ concentrations for Test Claimant 30, 10th June 2011

10/06/2011	Test Claimant 30			
Hour (hour ending BST)	Modelled concentrations including background (µg/m³)		Briery Hey background (µg/m³)	
	PM_{10}	$PM_{2.5}$	PM_{10}	$PM_{2.5}$
1	16.0	5.0	16.0	5.0
2	16.0	10.0	16.0	10.0
3	18.0	1.0	18.0	1.0
4	17.0	8.0	17.0	8.0
5	20.0	5.0	20.0	5.0
6	31.0	9.0	31.0	9.0
7	22.0	8.0	22.0	8.0
8	30.0	6.0	30.0	6.0
9	26.0	1.0	26.0	1.0
10	28.0	11.0	28.0	11.0
11	11.0	5.0	11.0	5.0
12	23.3	17.2	12.0	8.0
13	33.5	21.9	15.0	6.8
14	140.1	112.2	6.0	2.7
15	90.2	67.4	17.0	7.6
16	19.6	9.1	19.0	8.6
17	53.4	36.3	20.0	9.0
18	25.5	13.9	19.0	8.6
19	14.0	10.3	3.0	1.3
20	7.1	3.3	7.0	3.2
21	17.8	9.8	13.0	5.9
22	9.2	4.2	9.0	4.1
23	5.2	2.4	5.0	2.3
24	20.5	9.8	19.0	8.6
24-hour mean concentration (µg/m³)	**28.9**	**16.1**	***16.6***	***6.1***
24-hour mean concentration excluding background (µg/m³)	**12.3**	**10.1**		

Table 8: Modelled hourly PM_{10} and $PM_{2.5}$ concentrations for Test Claimant 30, 11th June 2011

11/06/2011	Test Claimant 30			
Hour (hour ending BST)	Modelled concentrations including background (µg/m³)		Briery Hey background (µg/m³)	
	PM_{10}	$PM_{2.5}$	PM_{10}	$PM_{2.5}$
1	17.0	7.7	17.0	7.7
2	8.0	3.6	8.0	3.6
3	11.0	5.0	11.0	4.9
4	18.1	8.2	18.0	8.1
5	18.0	8.1	18.0	8.1
6	15.0	6.8	15.0	6.7
7	19.1	8.6	19.0	8.6
8	26.0	12.1	25.0	11.2
9	28.1	12.7	28.0	12.6
10	19.6	9.0	19.0	8.6
11	15.0	7.8	12.0	5.4
12	20.1	9.1	20.0	9.0
13	20.6	9.5	20.0	9.0
14	22.1	12.2	16.0	7.2
15	12.6	5.9	12.0	5.4
16	26.8	15.6	17.0	7.6
17	16.3	8.1	14.0	6.3
18	12.2	5.5	12.0	5.4
19	28.5	19.6	10.0	4.5
20	17.2	8.5	15.0	6.8
21	15.5	8.3	12.0	5.4
22	16.1	9.1	11.0	5.0
23	11.0	5.0	11.0	5.0
24	12.0	5.4	12.0	5.4
24-hour mean concentration (µg/m³)	**17.7**	**8.8**	***15.5***	***7.0***
24-hour mean concentration excluding background (µg/m³)	**2.2**	**1.8**		

Table 9: Modelled hourly PM_{10} and $PM_{2.5}$ concentrations for Test Claimant 30, 27th June 2011

27/06/2011	Test Claimant 30			
Hour (hour ending BST)	Modelled concentrations including background (µg/m³)		Briery Hey background (µg/m³)	
	PM_{10}	$PM_{2.5}$	PM_{10}	$PM_{2.5}$
1	37.1	32.1	37.0	32.0
2	26.0	18.0	26.0	18.0
3	36.0	28.0	36.0	28.0
4	29.0	21.0	29.0	21.0
5	29.0	18.0	29.0	18.0
6	37.3	21.3	37.0	21.0
7	43.2	35.1	32.0	26.0
8	35.0	14.0	35.0	14.0
9	33.0	10.0	33.0	10.0
10	35.0	7.0	35.0	7.0
11	31.0	14.0	31.0	14.0
12	45.0	32.0	45.0	32.0
13	41.1	37.1	41.0	37.0
14	37.0	14.0	37.0	14.0
15	29.1	5.0	29.0	5.0
16	27.3	15.2	27.0	15.0
17	21.5	7.4	21.0	7.0
18	19.3	15.2	19.0	15.0
19	22.3	7.3	22.0	7.0
20	17.0	18.0	17.0	18.0
21	23.5	5.4	23.0	5.0
22	17.0	9.0	17.0	9.0
23	25.0	15.0	25.0	15.0
24	21.1	4.1	21.0	4.0
24-hour mean concentration (µg/m³)	**29.9**	**16.8**	***29.3***	***16.3***
24-hour mean concentration excluding background (µg/m³)	**0.6**	**0.5**		

Table 10: Modelled hourly PM_{10} and $PM_{2.5}$ concentrations for Test Claimant 33, 11th June 2011

11/06/2011	Test Claimant 33			
Hour (hour ending BST)	Modelled concentrations including background (µg/m³)		Briery Hey background (µg/m³)	
	PM_{10}	$PM_{2.5}$	PM_{10}	$PM_{2.5}$
1	17.0	7.7	17.0	7.7
2	8.0	3.6	8.0	3.6
3	11.0	5.0	11.0	4.9
4	18.0	8.1	18.0	8.1
5	31.2	18.9	18.0	8.1
6	15.0	6.8	15.0	6.7
7	19.1	8.6	19.0	8.6
8	25.1	11.3	25.0	11.2
9	29.8	14.1	28.0	12.6
10	19.8	9.2	19.0	8.6
11	12.4	5.8	12.0	5.4
12	36.5	22.5	20.0	9.0
13	24.7	12.8	20.0	9.0
14	16.1	7.3	16.0	7.2
15	13.0	6.2	12.0	5.4
16	17.0	7.7	17.0	7.7
17	14.4	6.6	14.0	6.3
18	14.6	7.6	12.0	5.4
19	10.0	4.5	10.0	4.5
20	15.0	6.8	15.0	6.8
21	12.0	5.4	12.0	5.4
22	11.0	5.0	11.0	4.9
23	11.0	5.0	11.0	4.9
24	12.0	5.4	12.0	5.4
24-hour mean concentration (µg/m³)	**17.2**	**8.4**	***15.5***	***7.0***
24-hour mean concentration excluding background (µg/m³)	**1.7**	**1.4**		

Table 11: Modelled hourly PM_{10} and $PM_{2.5}$ concentrations for Test Claimant 33, 15th June 2011

15/06/2011	Test Claimant 33			
Hour (hour ending BST)	Modelled concentrations including background (µg/m³)		Briery Hey background (µg/m³)	
	PM_{10}	$PM_{2.5}$	PM_{10}	$PM_{2.5}$
1	19.0	9.0	19.0	9.0
2	19.0	13.0	19.0	13.0
3	40.0	28.0	40.0	28.0
4	33.0	17.0	33.0	17.0
5	21.0	14.0	21.0	14.0
6	22.0	12.0	22.0	12.0
7	26.0	18.0	26.0	18.0
8	22.0	10.0	22.0	10.0
9	19.0	13.0	19.0	13.0
10	17.0	8.0	17.0	8.0
11	22.0	9.0	22.0	9.0
12	17.0	15.0	17.0	15.0
13	8.3	5.9	6.0	4.0
14	7.0	3.0	7.0	3.0
15	9.0	7.0	9.0	7.0
16	25.1	13.1	20.0	9.0
17	12.7	7.6	12.0	7.0
18	12.9	8.7	12.0	8.0
19	18.5	7.0	16.0	5.0
20	10.0	4.0	10.0	4.0
21	19.2	9.7	11.0	3.0
22	26.8	9.0	17.0	1.0
23	23.8	8.9	19.0	5.0
24	30.2	12.1	24.0	7.0
24-hour mean concentration (µg/m³)	**20.0**	**10.9**	***18.3***	***9.5***
24-hour mean concentration excluding background (µg/m³)	**1.7**	**1.4**		

Table 12: Modelled hourly PM_{10} and $PM_{2.5}$ concentrations for Test Claimant 33, 20th June 2011

20/06/2011	Test Claimant 33			
Hour (hour ending BST)	Modelled concentrations including background (µg/m³)		Briery Hey background (µg/m³)	
	PM_{10}	$PM_{2.5}$	PM_{10}	$PM_{2.5}$
1	43.7	30.2	19.0	10.0
2	44.0	28.6	20.0	9.0
3	22.0	18.0	22.0	18.0
4	21.8	9.1	18.0	6.0
5	17.7	9.6	17.0	9.0
6	21.3	8.3	21.0	8.0
7	21.0	15.0	21.0	15.0
8	41.0	2.0	41.0	2.0
9	32.0	5.0	32.0	5.0
10	18.6	7.5	18.0	7.0
11	26.1	5.1	26.0	5.0
12	21.7	6.6	21.0	6.0
13	16.4	9.3	16.0	9.0
14	21.8	10.7	21.0	10.0
15	21.4	25.2	20.0	24.0
16	19.2	16.0	18.0	15.0
17	12.0	5.0	12.0	5.0
18	15.0	13.0	15.0	13.0
19	17.0	16.0	17.0	16.0
20	21.0	10.0	21.0	10.0
21	19.0	19.0	19.0	19.0
22	31.0	15.0	31.0	15.0
23	25.0	17.0	25.0	17.0
24	32.0	22.0	32.0	22.0
24-hour mean concentration (µg/m³)	**24.2**	**13.5**	***21.8***	***11.5***
24-hour mean concentration excluding background (µg/m³)	**2.4**	**2.0**		

Table 13: Modelled hourly PM_{10} and $PM_{2.5}$ concentrations for Test Claimant 37, 10th June 2011

10/06/2011	Test Claimant 37			
Hour (hour ending BST)	Modelled concentrations including background (µg/m³)		Briery Hey background (µg/m³)	
	PM_{10}	$PM_{2.5}$	PM_{10}	$PM_{2.5}$
1	17.9	6.6	16.0	5.0
2	16.0	10.0	16.0	10.0
3	18.0	1.0	18.0	1.0
4	43.3	29.5	17.0	8.0
5	20.0	5.0	20.0	5.0
6	31.3	9.3	31.0	9.0
7	22.0	8.0	22.0	8.0
8	30.0	6.0	30.0	6.0
9	26.0	1.0	26.0	1.0
10	28.0	11.0	28.0	11.0
11	13.2	6.8	11.0	5.0
12	12.0	8.0	12.0	8.0
13	15.0	6.8	15.0	6.8
14	6.0	2.7	6.0	2.7
15	17.0	7.7	17.0	7.7
16	19.0	8.6	19.0	8.6
17	20.0	9.0	20.0	9.0
18	19.0	8.6	19.0	8.6
19	3.0	1.4	3.0	1.4
20	7.0	3.2	7.0	3.2
21	13.0	5.9	13.0	5.9
22	9.0	4.1	9.0	4.1
23	5.0	2.3	5.0	2.3
24	19.0	8.6	19.0	8.6
24-hour mean concentration (µg/m³)	**17.9**	**7.1**	***16.6***	***6.1***
24-hour mean concentration excluding background (µg/m³)	**1.3**	**1.0**		

Table 14: Modelled hourly PM_{10} and $PM_{2.5}$ concentrations for Test Claimant 37, 12th June 2011

12/06/2011	Test Claimant 37			
Hour (hour ending BST)	Modelled concentrations including background (µg/m³)		Briery Hey background (µg/m³)	
	PM_{10}	$PM_{2.5}$	PM_{10}	$PM_{2.5}$
1	16.0	7.2	16.0	7.2
2	21.0	9.5	21.0	9.5
3	20.0	9.0	20.0	9.0
4	18.0	8.1	18.0	8.1
5	21.0	9.5	21.0	9.5
6	20.0	9.0	20.0	9.0
7	41.6	24.1	27.0	12.2
8	87.3	46.9	36.0	5.0
9	54.7	27.5	32.0	9.0
10	128.3	88.5	26.0	5.0
11	135.8	96.4	30.0	10.0
12	119.9	86.6	15.0	1.0
13	72.2	60.9	11.0	11.0
14	89.5	65.3	23.0	11.0
15	44.4	30.5	18.0	9.0
16	51.8	41.8	14.0	11.0
17	29.9	12.3	16.0	1.0
18	94.8	62.7	18.0	0.0
19	94.9	73.0	14.0	7.0
20	104.7	79.1	20.0	10.0
21	111.6	84.4	18.0	8.0
22	122.3	90.6	26.0	12.0
23	64.8	49.8	16.0	10.0
24	10.2	7.2	10.0	7.0
24-hour mean concentration (µg/m³)	**65.6**	**45.0**	***20.3***	***8.0***
24-hour mean concentration excluding background (µg/m³)	**45.4**	**37.0**		

Table 15: Modelled hourly PM_{10} and $PM_{2.5}$ concentrations for Test Claimant 37, 17th June 2011

17/06/2011	Test Claimant 37			
Hour (hour ending BST)	Modelled concentrations including background (µg/m³)		Briery Hey background (µg/m³)	
	PM_{10}	$PM_{2.5}$	PM_{10}	$PM_{2.5}$
1	26.0	10.0	26.0	10.0
2	22.0	18.0	22.0	18.0
3	20.0	10.0	20.0	10.0
4	24.0	7.0	24.0	7.0
5	24.0	12.0	24.0	12.0
6	27.0	12.0	27.0	12.0
7	31.0	11.3	27.0	8.0
8	23.5	7.2	22.0	6.0
9	31.2	17.9	24.0	12.0
10	41.1	17.9	40.0	17.0
11	32.7	13.2	30.0	11.0
12	85.6	11.3	84.0	10.0
13	44.0	12.8	43.0	12.0
14	81.7	15.2	79.0	13.0
15	64.7	6.4	63.0	5.0
16	100.9	13.7	100.0	13.0
17	27.5	6.0	25.0	4.0
18	21.5	8.3	20.0	7.0
19	17.8	2.6	17.0	2.0
20	18.3	12.7	15.0	10.0
21	28.0	13.1	18.0	5.0
22	27.3	13.6	18.0	6.0
23	31.6	21.8	22.0	14.0
24	31.6	32.8	22.0	25.0
24-hour mean concentration (µg/m³)	**36.8**	**12.8**	***33.8***	***10.4***
24-hour mean concentration excluding background (µg/m³)	**3.0**	**2.4**		

Table 16: Modelled hourly PM_{10} and $PM_{2.5}$ concentrations for Test Claimant 39, 10th June 2011

10/06/2011	Test Claimant 39			
Hour (hour ending BST)	Modelled concentrations including background (µg/m³)		Briery Hey background (µg/m³)	
	PM_{10}	$PM_{2.5}$	PM_{10}	$PM_{2.5}$
1	16.0	5.0	16.0	5.0
2	16.0	10.0	16.0	10.0
3	18.0	1.0	18.0	1.0
4	17.0	8.0	17.0	8.0
5	20.0	5.0	20.0	5.0
6	35.5	12.7	31.0	9.0
7	22.0	8.0	22.0	8.0
8	30.1	6.1	30.0	6.0
9	26.4	1.3	26.0	1.0
10	28.0	11.0	28.0	11.0
11	19.0	11.5	11.0	5.0
12	12.0	8.0	12.0	8.0
13	15.0	6.8	15.0	6.8
14	6.0	2.7	6.0	2.7
15	17.0	7.7	17.0	7.7
16	19.0	8.6	19.0	8.6
17	20.0	9.0	20.0	9.0
18	19.0	8.6	19.0	8.6
19	3.0	1.4	3.0	1.4
20	7.0	3.2	7.0	3.2
21	13.0	5.9	13.0	5.9
22	9.0	4.1	9.0	4.1
23	5.0	2.3	5.0	2.3
24	19.0	8.6	19.0	8.6
24-hour mean concentration (µg/m³)	**17.2**	**6.5**	***16.6***	***6.1***
24-hour mean concentration excluding background (µg/m³)	**0.5**	**0.4**		

Table 17: Modelled hourly PM_{10} and $PM_{2.5}$ concentrations for Test Claimant 39, 12th June 2011

12/06/2011	Test Claimant 39			
Hour (hour ending BST)	Modelled concentrations including background (µg/m³)		Briery Hey background (µg/m³)	
	PM_{10}	$PM_{2.5}$	PM_{10}	$PM_{2.5}$
1	16.0	7.2	16.0	7.2
2	21.0	9.5	21.0	9.5
3	20.0	9.0	20.0	9.0
4	18.0	8.1	18.0	8.1
5	21.0	9.5	21.0	9.5
6	21.2	10.0	20.0	9.0
7	27.0	12.2	27.0	12.1
8	36.0	5.0	36.0	5.0
9	32.0	9.0	32.0	9.0
10	27.0	5.8	26.0	5.0
11	31.8	11.5	30.0	10.0
12	19.5	4.7	15.0	1.0
13	11.1	11.1	11.0	11.0
14	23.3	11.2	23.0	11.0
15	18.0	9.0	18.0	9.0
16	14.0	11.0	14.0	11.0
17	16.0	1.0	16.0	1.0
18	18.7	0.5	18.0	0.0
19	20.9	12.6	14.0	7.0
20	21.2	11.0	20.0	10.0
21	20.1	9.7	18.0	8.0
22	29.7	15.0	26.0	12.0
23	32.3	23.3	16.0	10.0
24	24.5	18.9	10.0	7.0
24-hour mean concentration (µg/m³)	**22.5**	**9.8**	***20.2***	***8.0***
24-hour mean concentration excluding background (µg/m³)	**2.3**	**1.9**		

Table 18: Modelled hourly PM_{10} and $PM_{2.5}$ concentrations for Test Claimant 39, 17th June 2011

17/06/2011	Test Claimant 39			
Hour (hour ending BST)	Modelled concentrations including background (µg/m³)		Briery Hey background (µg/m³)	
	PM_{10}	$PM_{2.5}$	PM_{10}	$PM_{2.5}$
1	26.0	10.0	26.0	10.0
2	22.0	18.0	22.0	18.0
3	25.0	14.1	20.0	10.0
4	24.1	7.1	24.0	7.0
5	24.0	12.0	24.0	12.0
6	27.3	12.3	27.0	12.0
7	28.8	9.5	27.0	8.0
8	23.6	7.3	22.0	6.0
9	24.2	12.2	24.0	12.0
10	41.1	17.9	40.0	17.0
11	31.0	11.8	30.0	11.0
12	85.0	10.9	84.0	10.0
13	44.0	12.8	43.0	12.0
14	80.0	13.8	79.0	13.0
15	64.0	5.8	63.0	5.0
16	101.0	13.8	100.0	13.0
17	26.0	4.8	25.0	4.0
18	21.3	8.1	20.0	7.0
19	18.5	3.2	17.0	2.0
20	16.5	11.2	15.0	10.0
21	18.2	5.2	18.0	5.0
22	18.6	6.5	18.0	6.0
23	22.1	14.1	22.0	14.0
24	22.2	25.2	22.0	25.0
24-hour mean concentration (µg/m³)	**34.8**	**11.2**	***33.8***	***10.4***
24-hour mean concentration excluding background (µg/m³)	**0.9**	**0.8**		

Sonae GLO: Further Modelling

4. Histograms of modelled hourly concentrations for the 20 Test Claimants

i) *Unit discharge concentrations with equivalent thresholds for acrolein and total aldehydes*

This document presents time series of hourly average concentrations for a 'unit discharge' at the locations of the 20 Test Claimants.

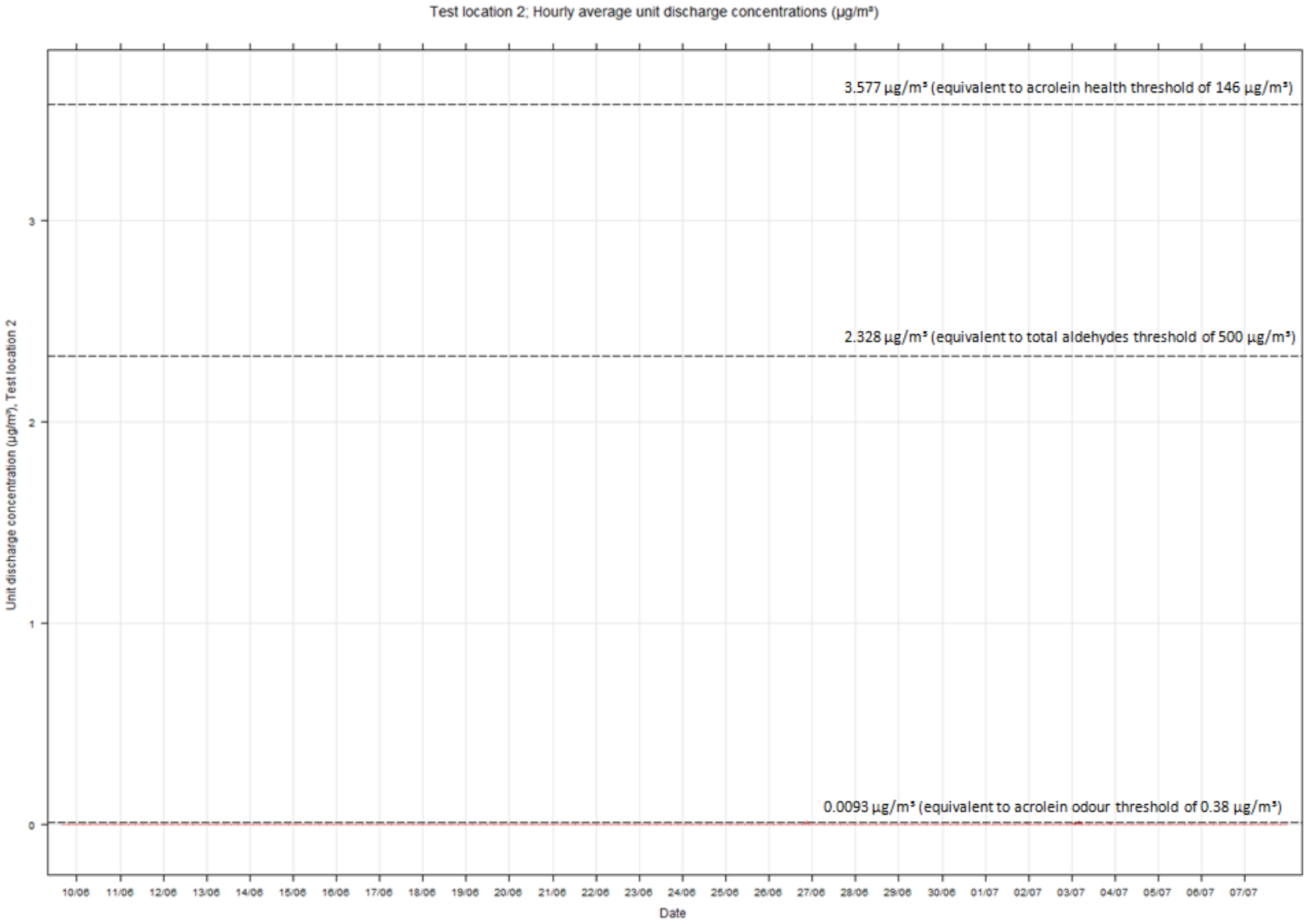
The 'unit discharge' rate of 1 g/s is used for peak heat release rate of 358 MW, equivalent to an emission factor of 2.793 mg/s/MW. Since the emission factors for acrolein and total aldehydes are assumed to be constant throughout the lifetime of the fire, thresholds equivalent to the exceedence thresholds for these pollutants can be displayed on the unit discharge time series.

The equivalent thresholds are derived using the following formula:

$$\frac{\text{unit discharge emission factor (2.793 mg/s/MW)}}{\text{pollutant emission factor (mg/s/MW)}} \times \text{pollutant exceedence threshold } (\mu g/m^3)$$

The table below shows the equivalent thresholds for acrolein and total aldehydes based on emission factors of 114 mg/s/MW and 600 mg/s/MW, respectively. These equivalent threshold values are also shown in the following 23 time series histograms for the locations of the 20 Test Claimants.

Pollutant	**Emission Factor (mg/s/MW)**	**Pollutant exceedence threshold (µg/m³)**	**Equivalent threshold for unit discharge modelling (µg/m³)**
Acrolein	114	0.38 *(odour threshold)*	0.0093
		146 *(health threshold)*	3.577
Total aldehydes	600	500	2.328


Test location 2; Hourly average unit discharge concentrations (µg/m³)
3.577 µg/m³ (equivalent to acrolein health threshold of 146 µg/m³)
2.328 µg/m³ (equivalent to total aldehydes threshold of 500 µg/m³)
0.0093 µg/m³ (equivalent to acrolein odour threshold of 0.38 µg/m³)
Unit discharge concentration (µg/m³), Test location 2
0
1
2
3
10/06
11/06
12/06
13/06
14/06
15/06
16/06
17/06
18/06
19/06
20/06
21/06
22/06
23/06
24/06
25/06
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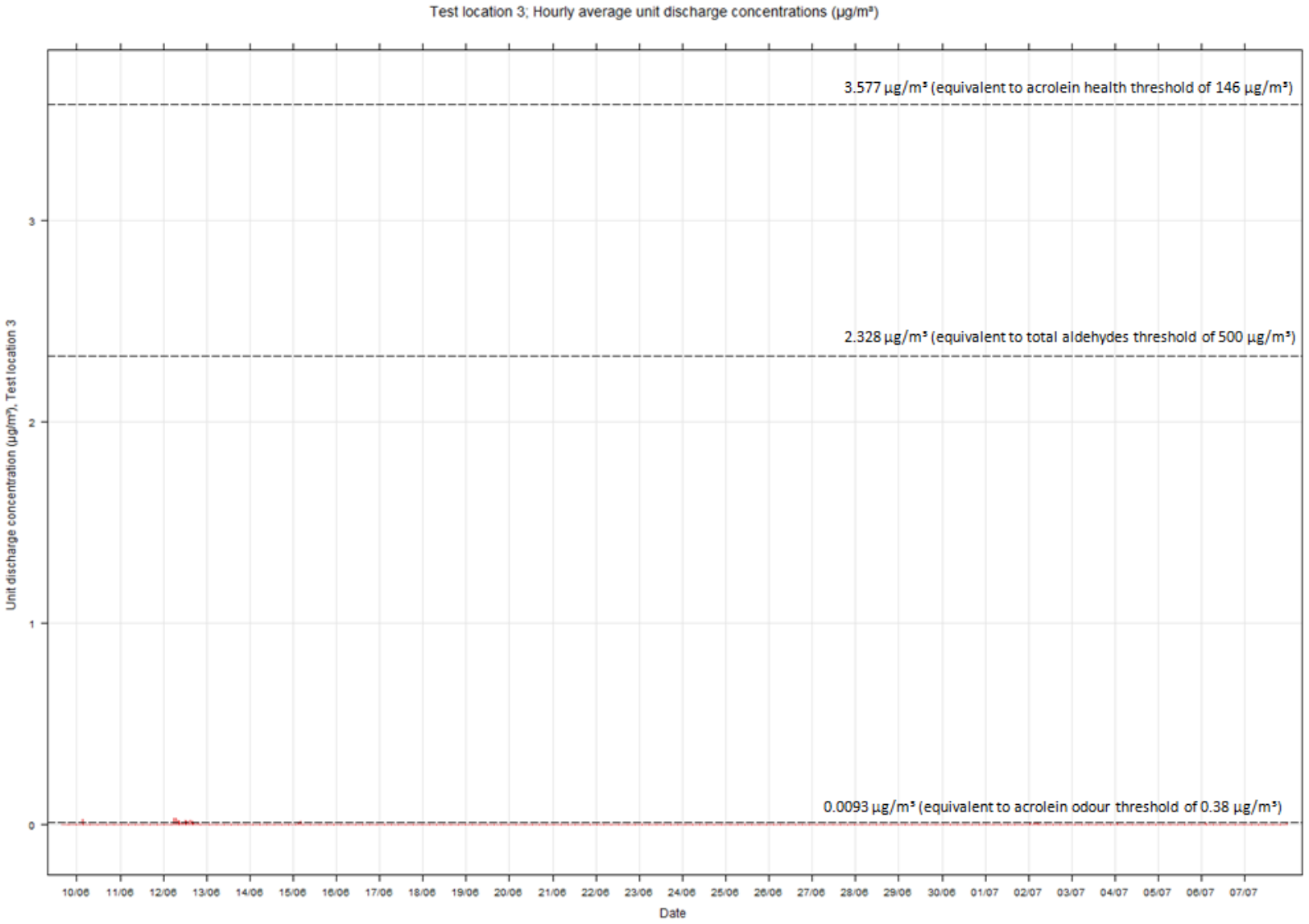
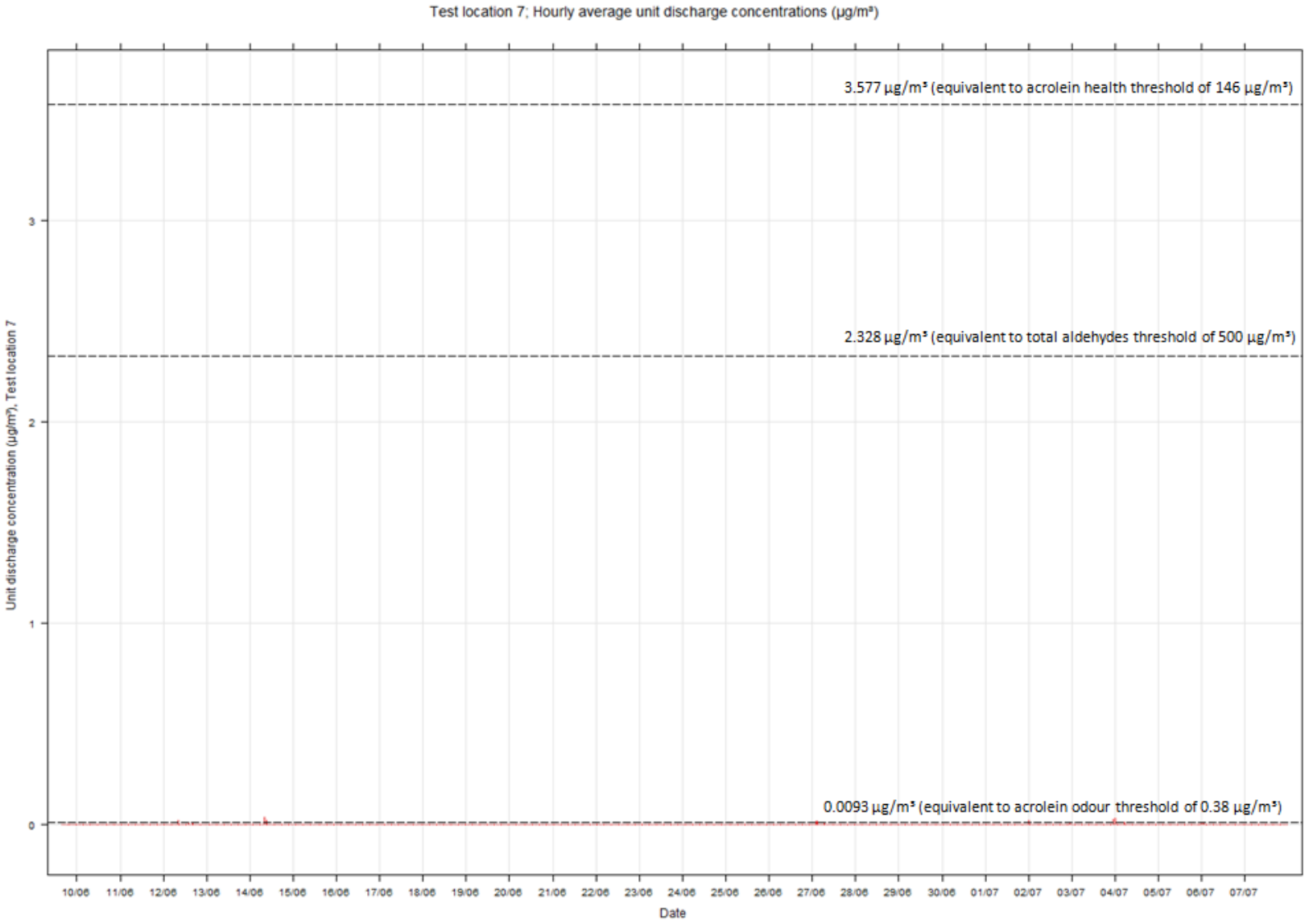
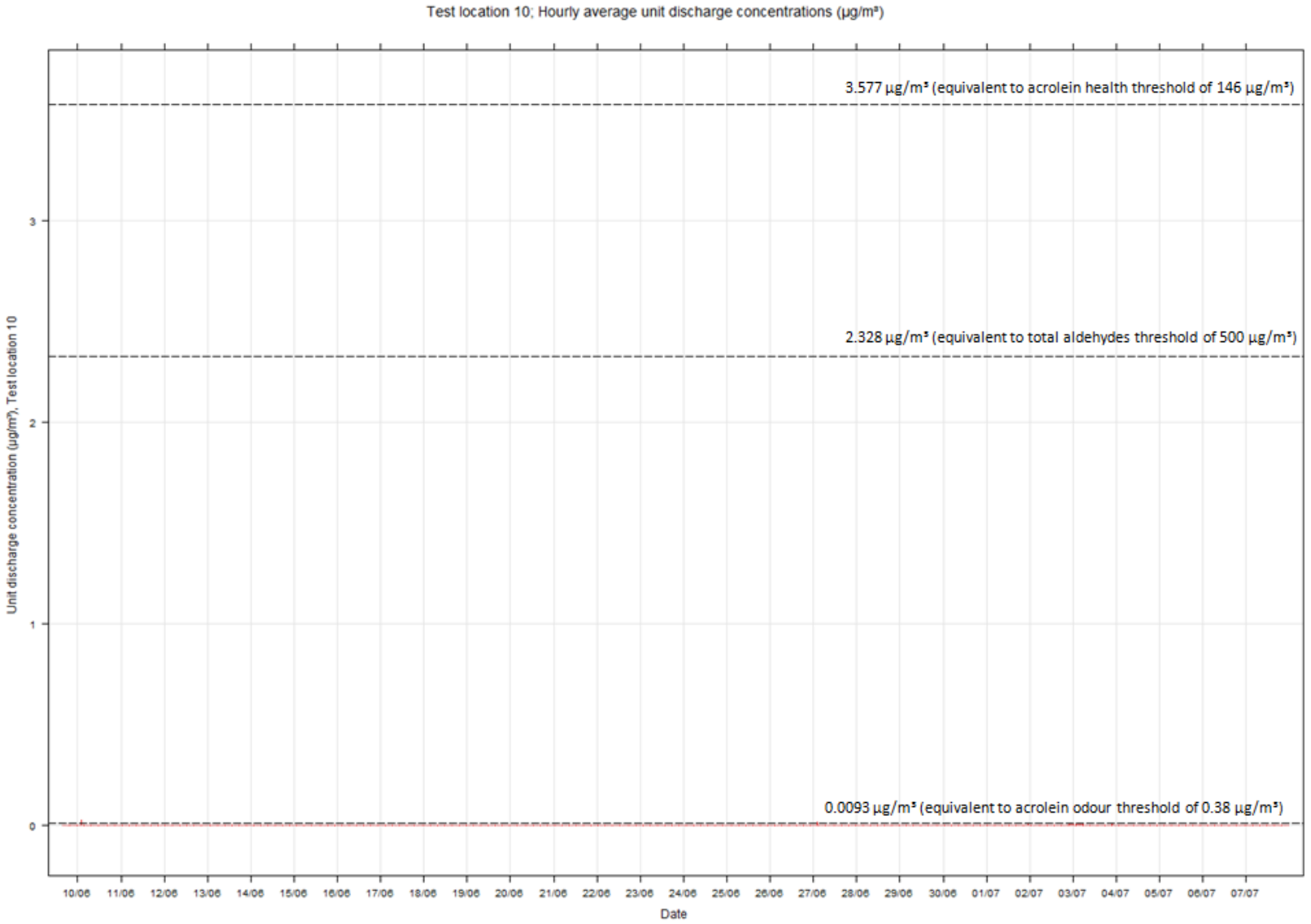
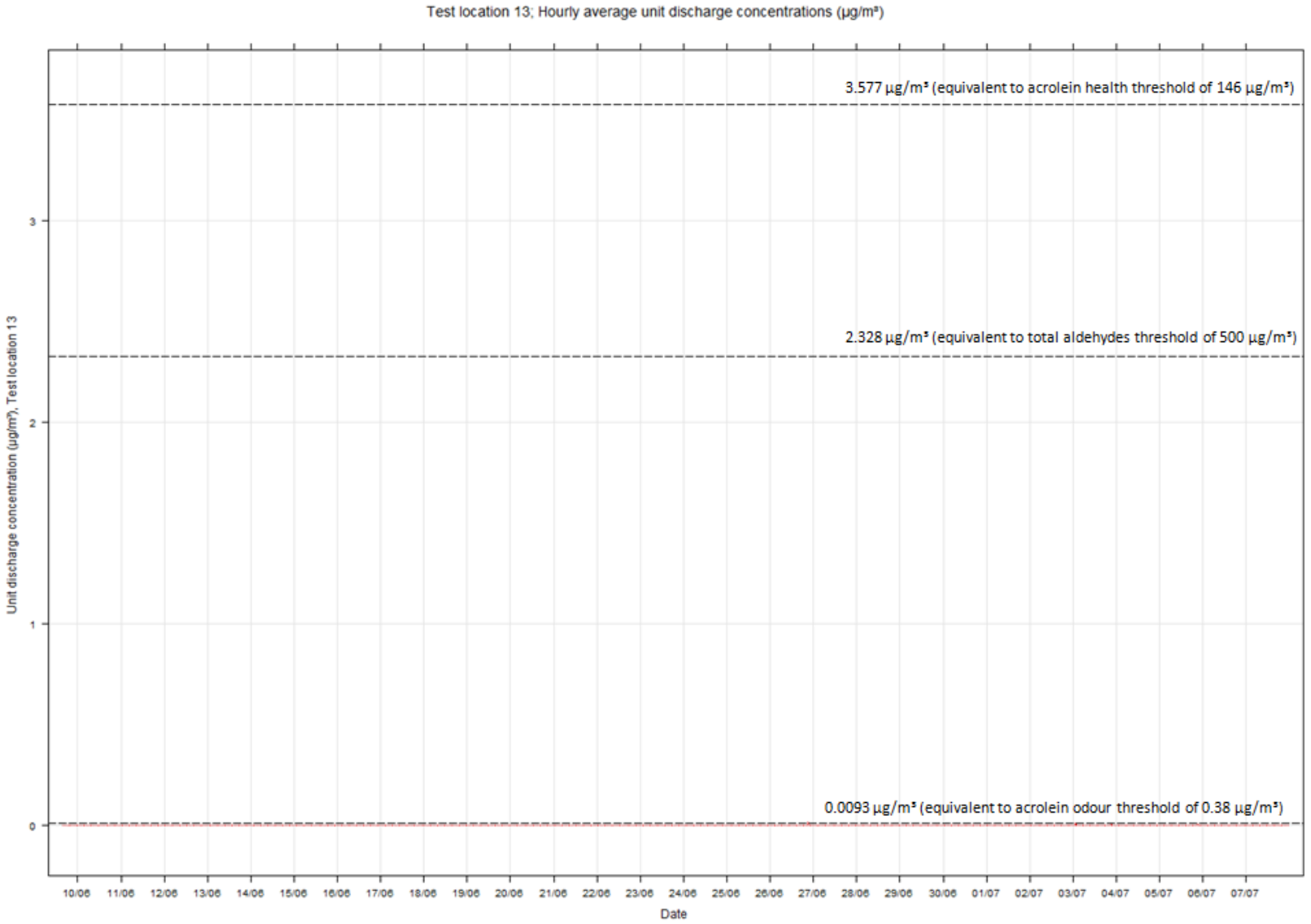
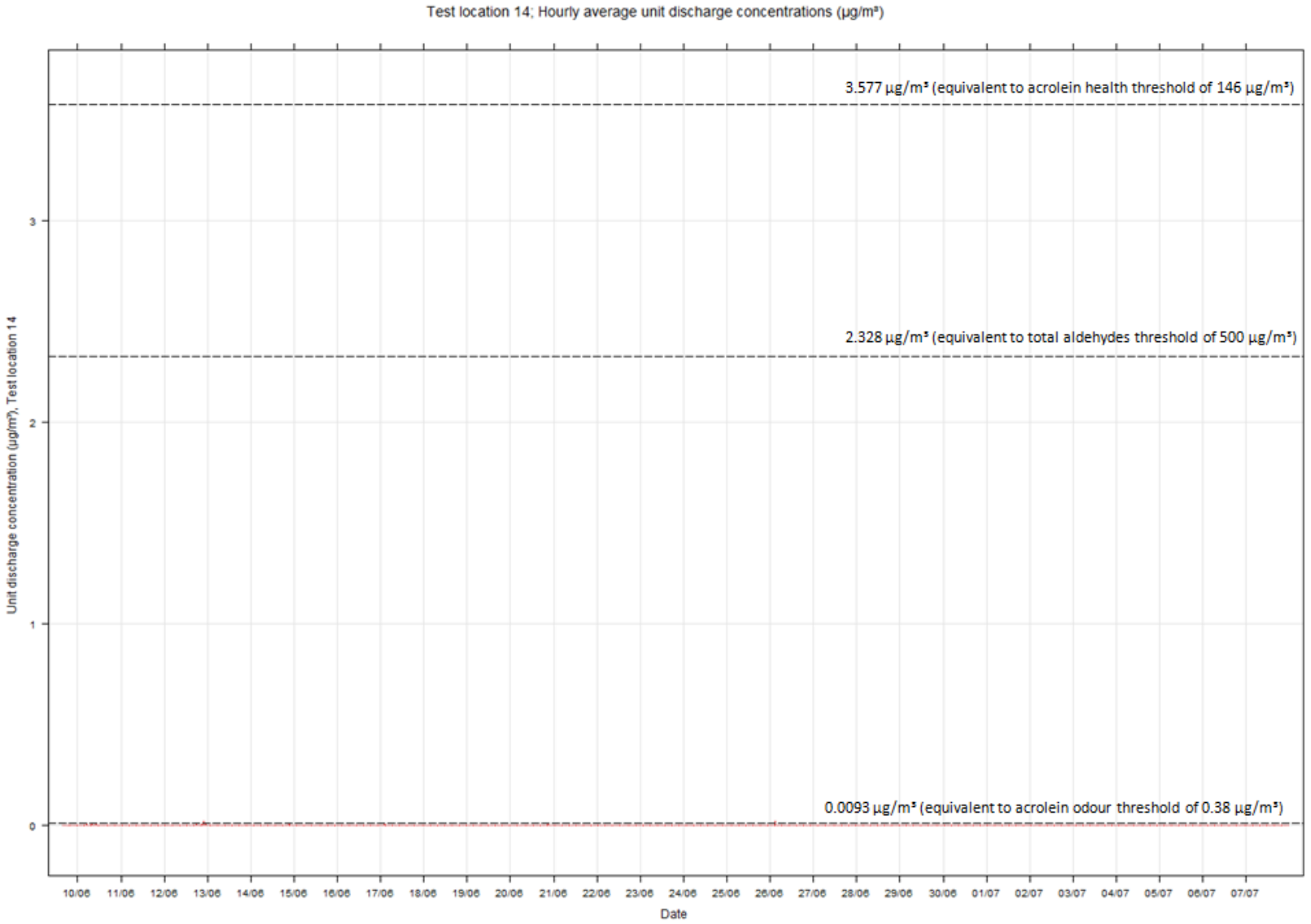
03/07
04/07
05/07
06/07
07/07
Date

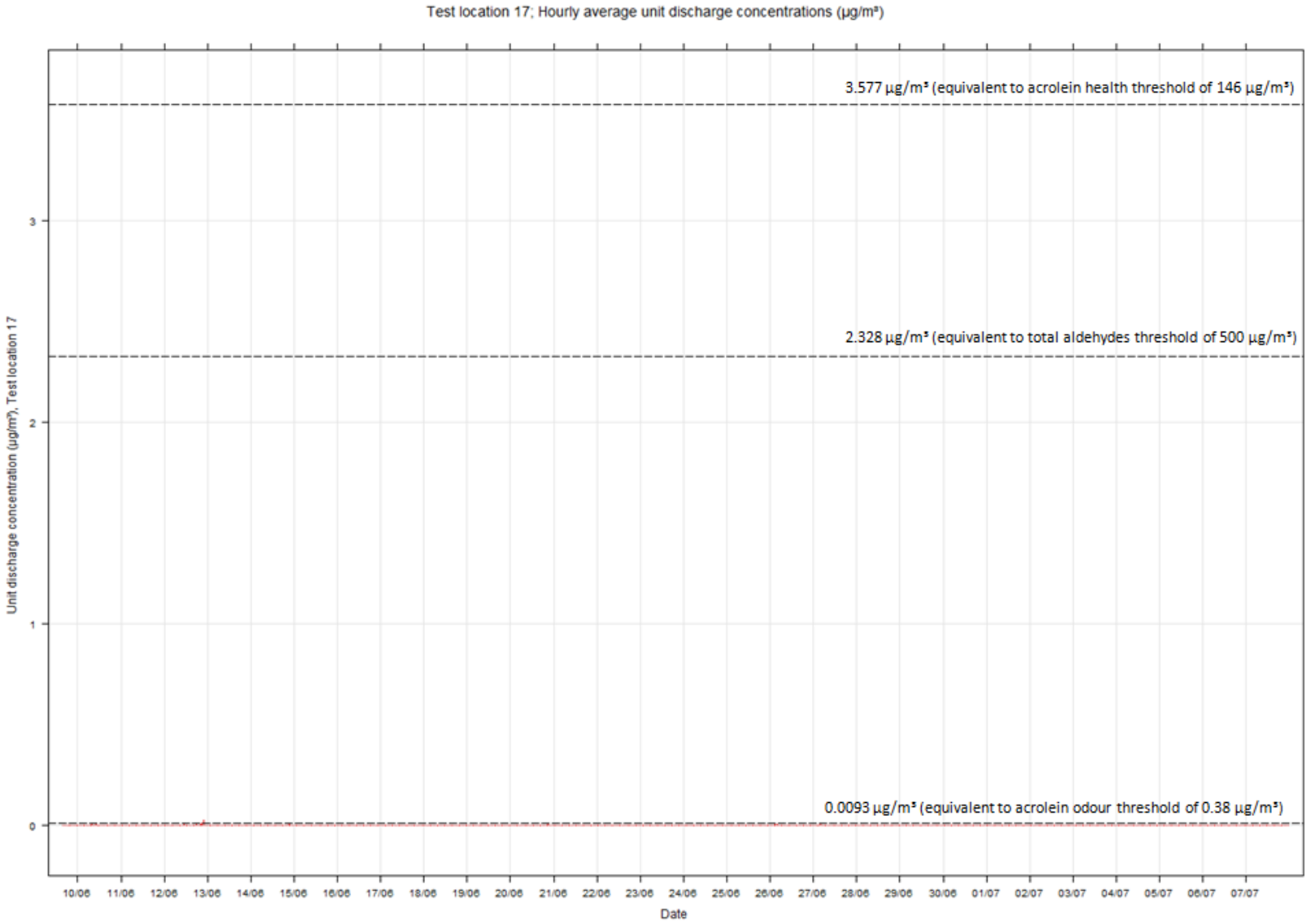

Test location 3; Hourly average unit discharge concentrations (µg/m³)
3.577 µg/m³ (equivalent to acrolein health threshold of 146 µg/m³)
2.328 µg/m³ (equivalent to total aldehydes threshold of 500 µg/m³)
0.0093 µg/m³ (equivalent to acrolein odour threshold of 0.38 µg/m³)
Unit discharge concentration (µg/m³), Test location 3
0
1
2
3
10/06
11/06
12/06
13/06
14/06
15/06
16/06
17/06
18/06
19/06
20/06
21/06
22/06
23/06
24/06
25/06
26/06
27/06
28/06
29/06
30/06
01/07
02/07
03/07
04/07
05/07
06/07
07/07
Date

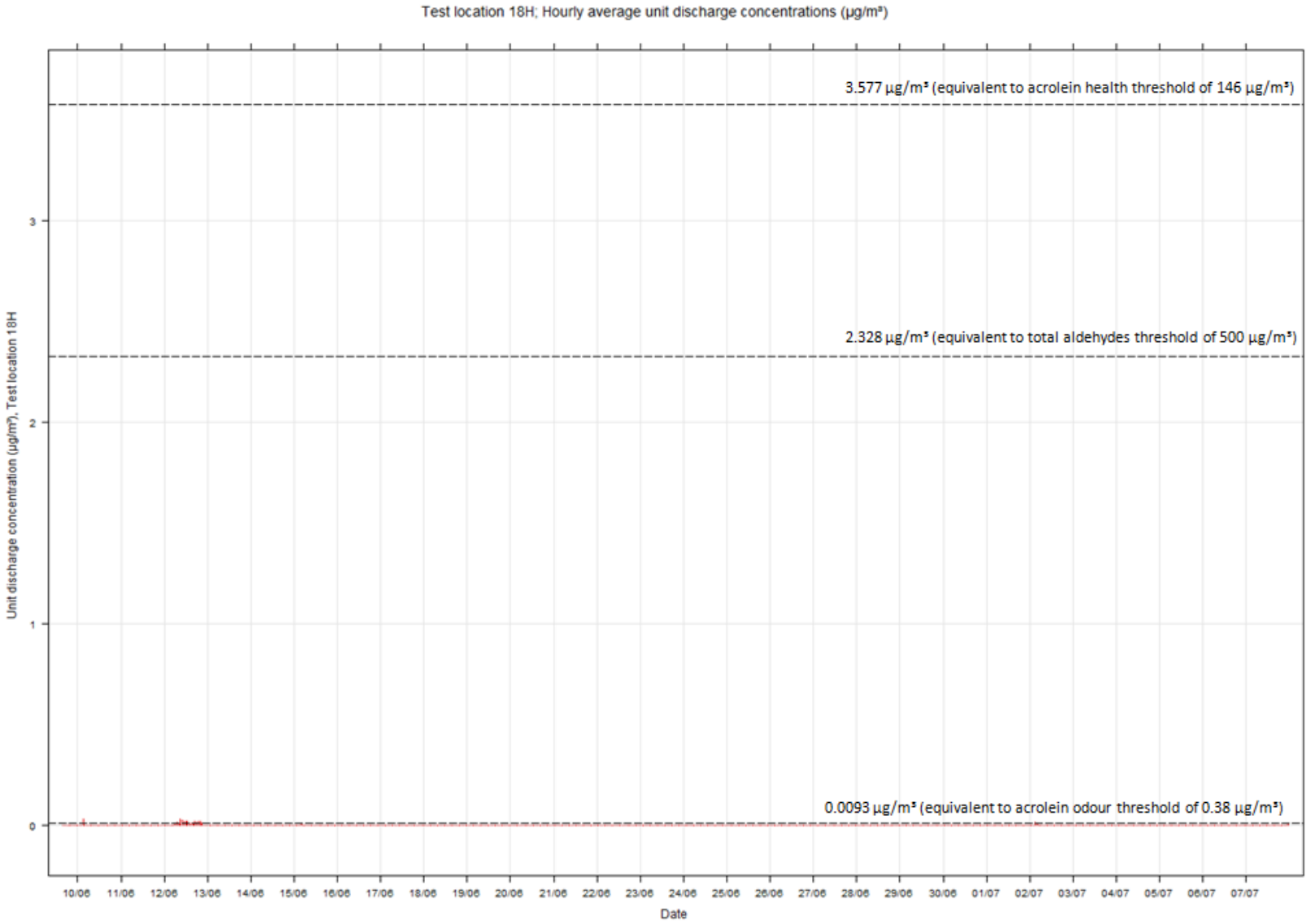

Test location 7; Hourly average unit discharge concentrations (µg/m³)
3.577 µg/m³ (equivalent to acrolein health threshold of 146 µg/m³)
2.328 µg/m³ (equivalent to total aldehydes threshold of 500 µg/m³)
0.0093 µg/m³ (equivalent to acrolein odour threshold of 0.38 µg/m³)
Unit discharge concentration (µg/m³), Test location 7
0
1
2
3
10/06
11/06
12/06
13/06
14/06
15/06
16/06
17/06
18/06
19/06
20/06
21/06
22/06
23/06
24/06
25/06
26/06
27/06
28/06
29/06
30/06
01/07
02/07
03/07
04/07
05/07
06/07
07/07
Date

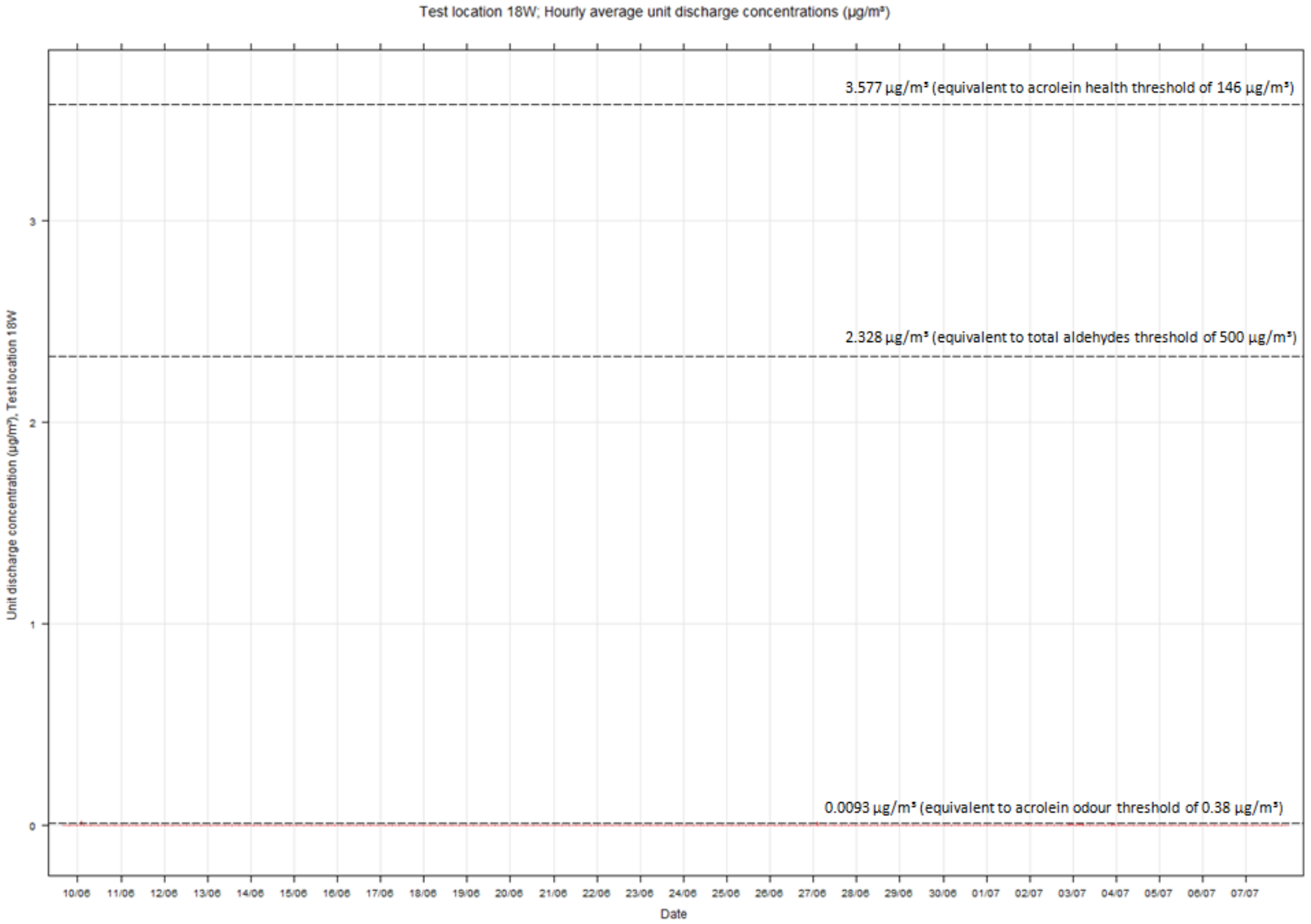

Test location 10; Hourly average unit discharge concentrations (µg/m³)
3.577 µg/m³ (equivalent to acrolein health threshold of 146 µg/m³)
2.328 µg/m³ (equivalent to total aldehydes threshold of 500 µg/m³)
0.0093 µg/m³ (equivalent to acrolein odour threshold of 0.38 µg/m³)
Unit discharge concentration (µg/m³), Test location 10
0
1
2
3
10/06 11/06 12/06 13/06 14/06 15/06 16/06 17/06 18/06 19/06 20/06 21/06 22/06 23/06 24/06 25/06 26/06 27/06 28/06 29/06 30/06 01/07 02/07 03/07 04/07 05/07 06/07 07/07
Date

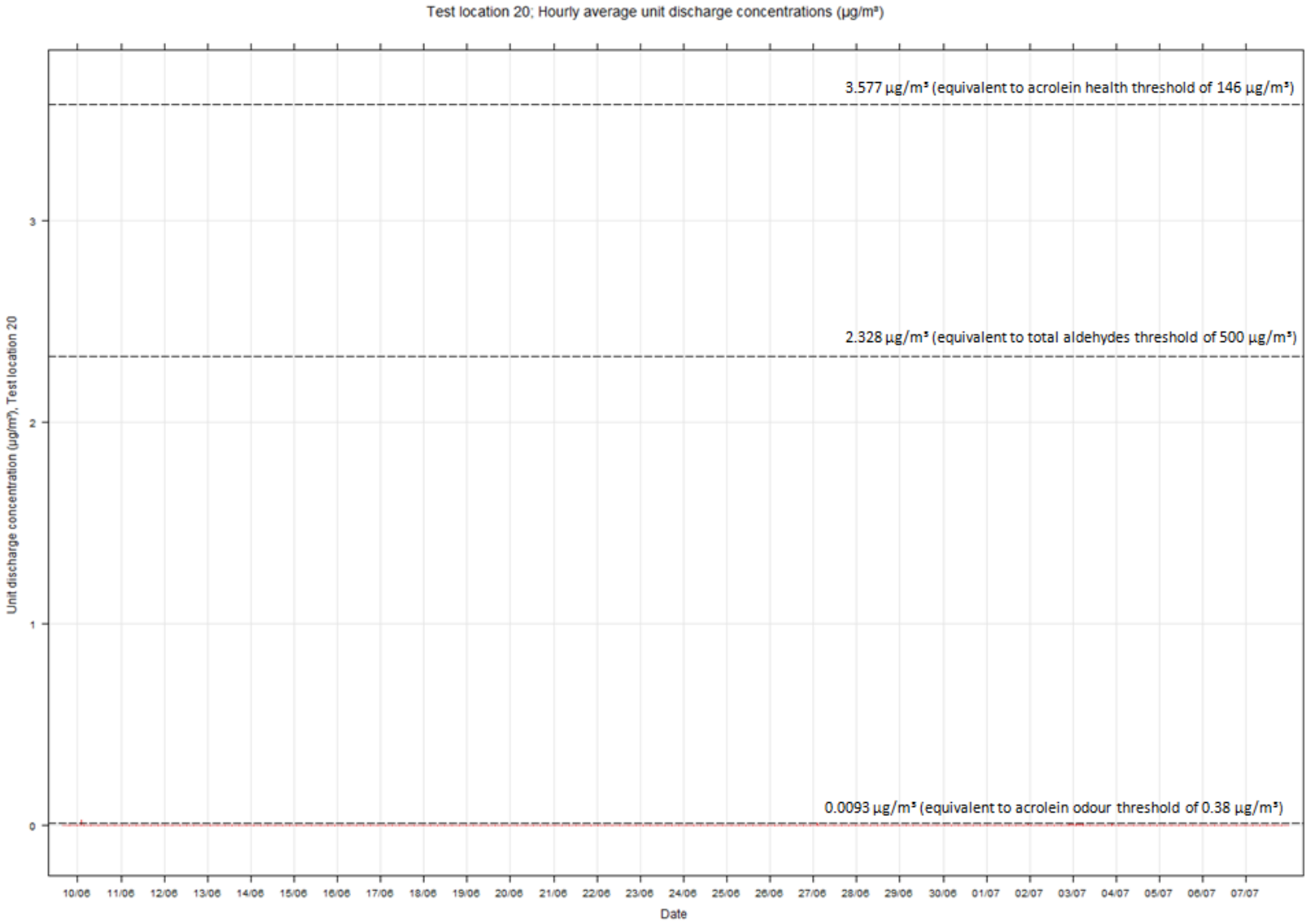

Test location 13; Hourly average unit discharge concentrations (µg/m³)
3.577 µg/m³ (equivalent to acrolein health threshold of 146 µg/m³)
2.328 µg/m³ (equivalent to total aldehydes threshold of 500 µg/m³)
0.0093 µg/m³ (equivalent to acrolein odour threshold of 0.38 µg/m³)
Unit discharge concentration (µg/m³), Test location 13
0
1
2
3
10/06
11/06
12/06
13/06
14/06
15/06
16/06
17/06
18/06
19/06
20/06
21/06
22/06
23/06
24/06
25/06
26/06
27/06
28/06
29/06
30/06
01/07
02/07
03/07
04/07
05/07
06/07
07/07
Date

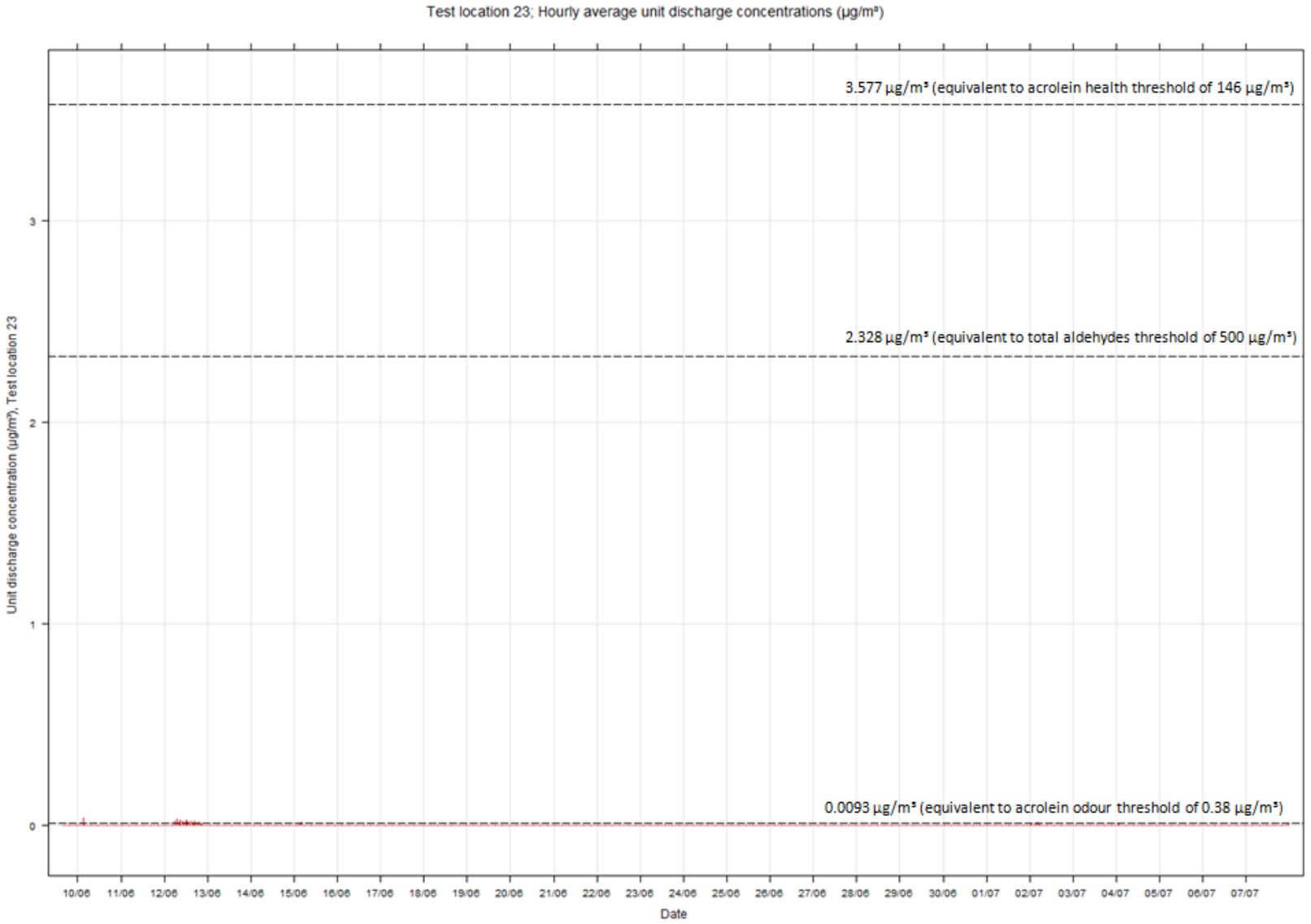

Test location 14; Hourly average unit discharge concentrations (µg/m³)
3.577 µg/m³ (equivalent to acrolein health threshold of 146 µg/m³)
2.328 µg/m³ (equivalent to total aldehydes threshold of 500 µg/m³)
0.0093 µg/m³ (equivalent to acrolein odour threshold of 0.38 µg/m³)
Unit discharge concentration (µg/m³), Test location 14
0
1
2
3
10/06 11/06 12/06 13/06 14/06 15/06 16/06 17/06 18/06 19/06 20/06 21/06 22/06 23/06 24/06 25/06 26/06 27/06 28/06 29/06 30/06 01/07 02/07 03/07 04/07 05/07 06/07 07/07
Date

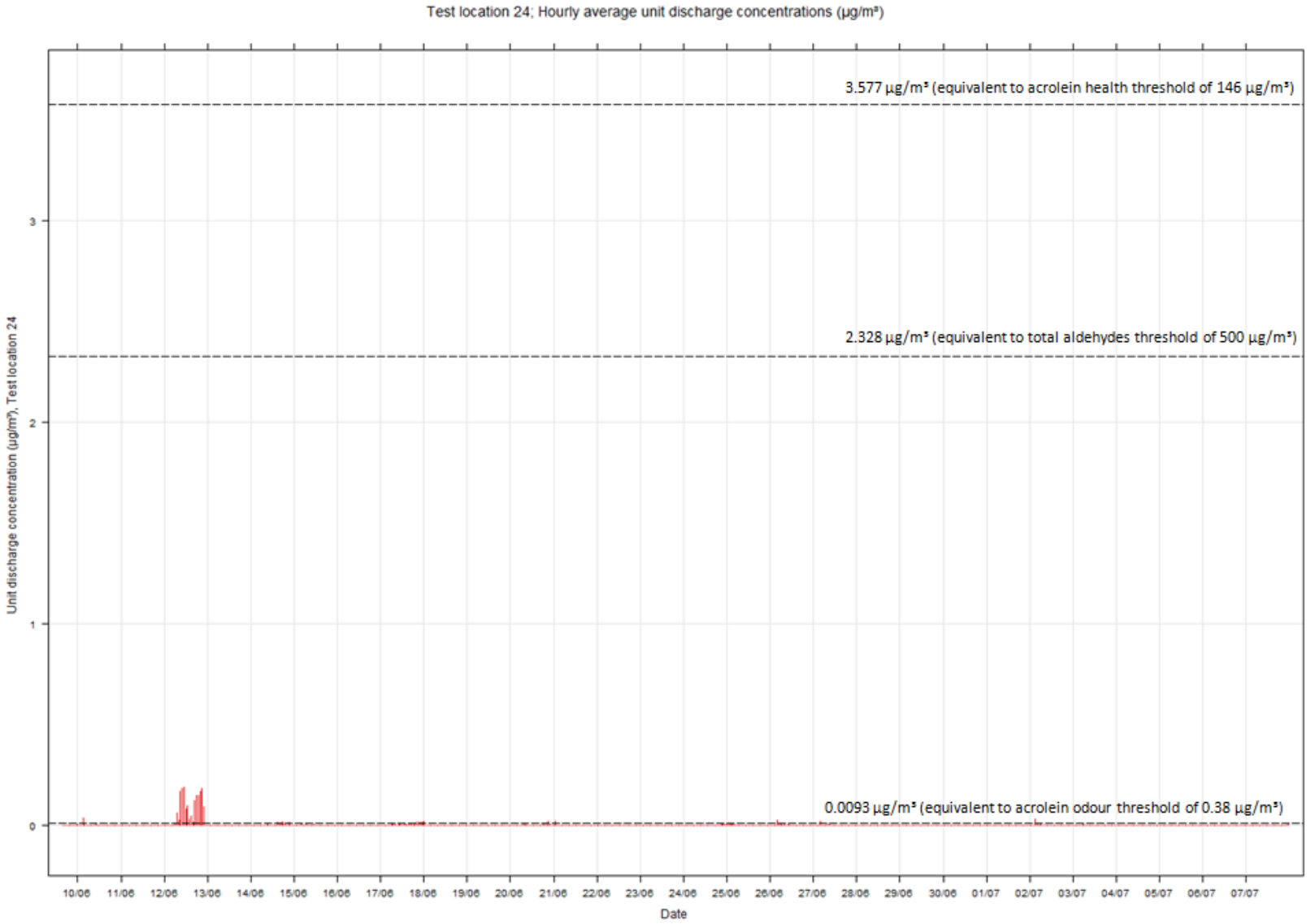

Test location 17; Hourly average unit discharge concentrations (µg/m³)
3.577 µg/m³ (equivalent to acrolein health threshold of 146 µg/m³)
2.328 µg/m³ (equivalent to total aldehydes threshold of 500 µg/m³)
0.0093 µg/m³ (equivalent to acrolein odour threshold of 0.38 µg/m³)
Unit discharge concentration (µg/m³), Test location 17
0
1
2
3
10/06 11/06 12/06 13/06 14/06 15/06 16/06 17/06 18/06 19/06 20/06 21/06 22/06 23/06 24/06 25/06 26/06 27/06 28/06 29/06 30/06 01/07 02/07 03/07 04/07 05/07 06/07 07/07
Date

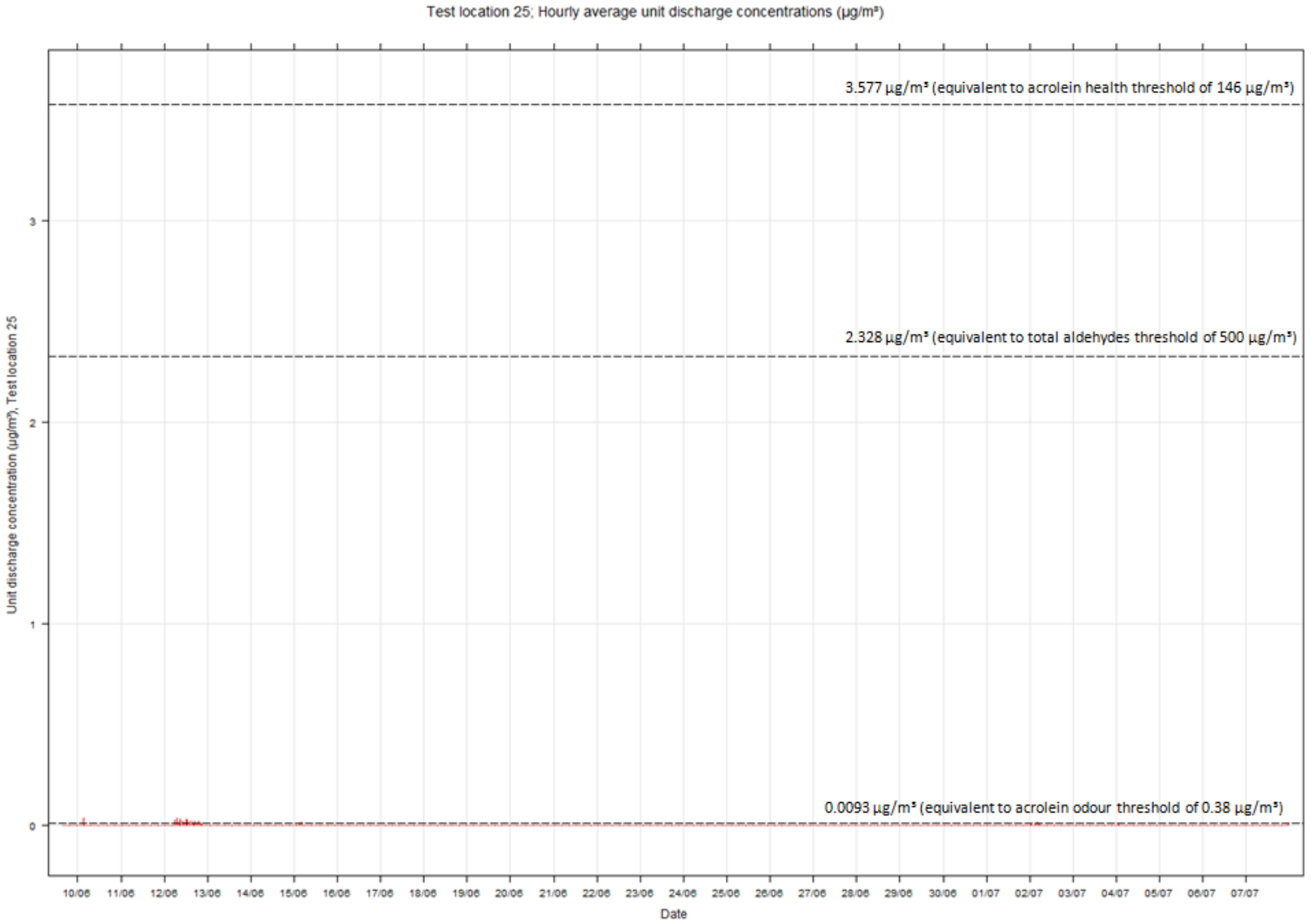

Test location 18H; Hourly average unit discharge concentrations (µg/m³)
3.577 µg/m³ (equivalent to acrolein health threshold of 146 µg/m³)
2.328 µg/m³ (equivalent to total aldehydes threshold of 500 µg/m³)
0.0093 µg/m³ (equivalent to acrolein odour threshold of 0.38 µg/m³)
Unit discharge concentration (µg/m³), Test location 18H
0
1
2
3
10/06 11/06 12/06 13/06 14/06 15/06 16/06 17/06 18/06 19/06 20/06 21/06 22/06 23/06 24/06 25/06 26/06 27/06 28/06 29/06 30/06 01/07 02/07 03/07 04/07 05/07 06/07 07/07
Date

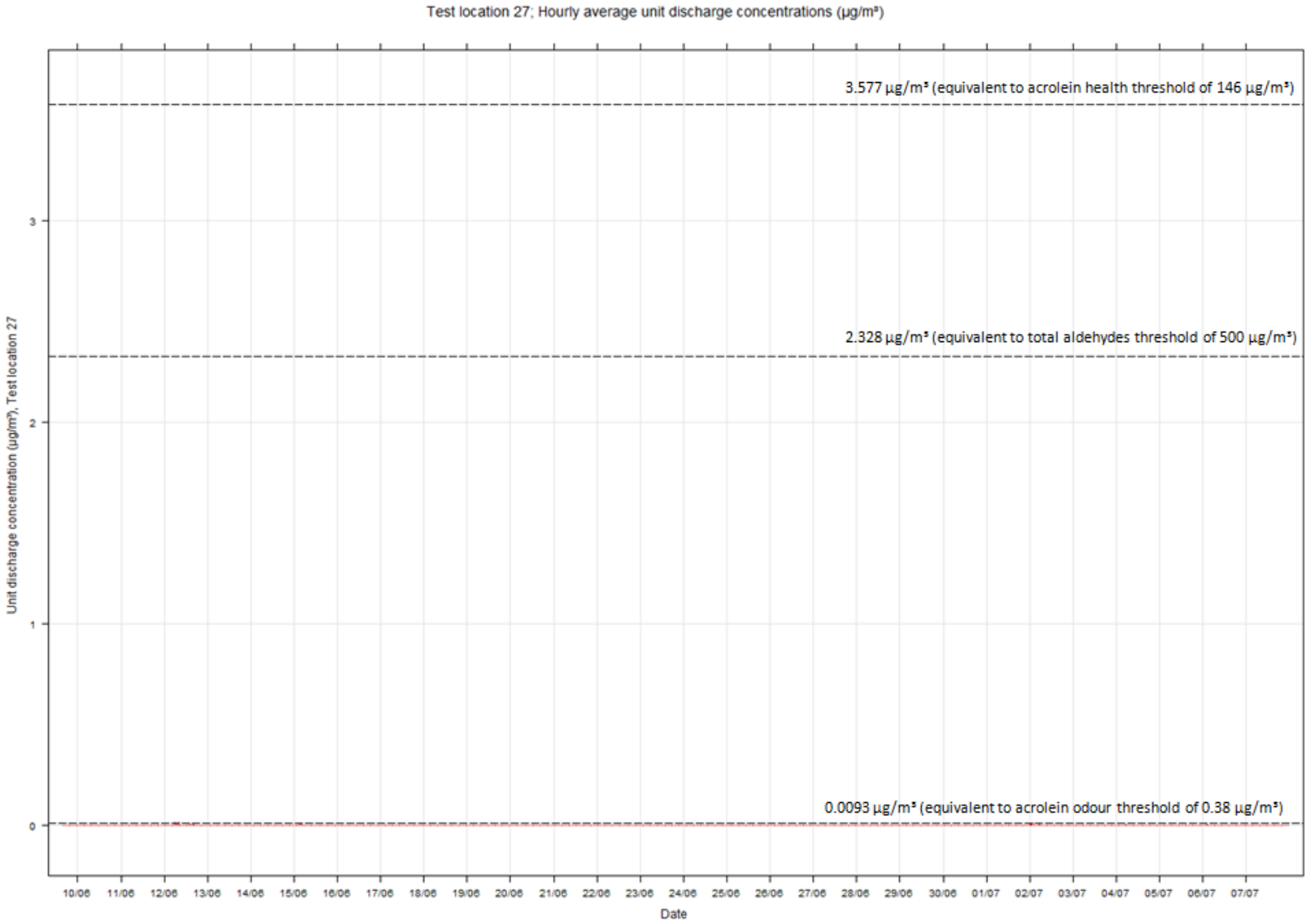

Test location 18W; Hourly average unit discharge concentrations (µg/m³)
3.577 µg/m³ (equivalent to acrolein health threshold of 146 µg/m³)
2.328 µg/m³ (equivalent to total aldehydes threshold of 500 µg/m³)
0.0093 µg/m³ (equivalent to acrolein odour threshold of 0.38 µg/m³)
Unit discharge concentration (µg/m³), Test location 18W
0
1
2
3
10/06 11/06 12/06 13/06 14/06 15/06 16/06 17/06 18/06 19/06 20/06 21/06 22/06 23/06 24/06 25/06 26/06 27/06 28/06 29/06 30/06 01/07 02/07 03/07 04/07 05/07 06/07 07/07
Date

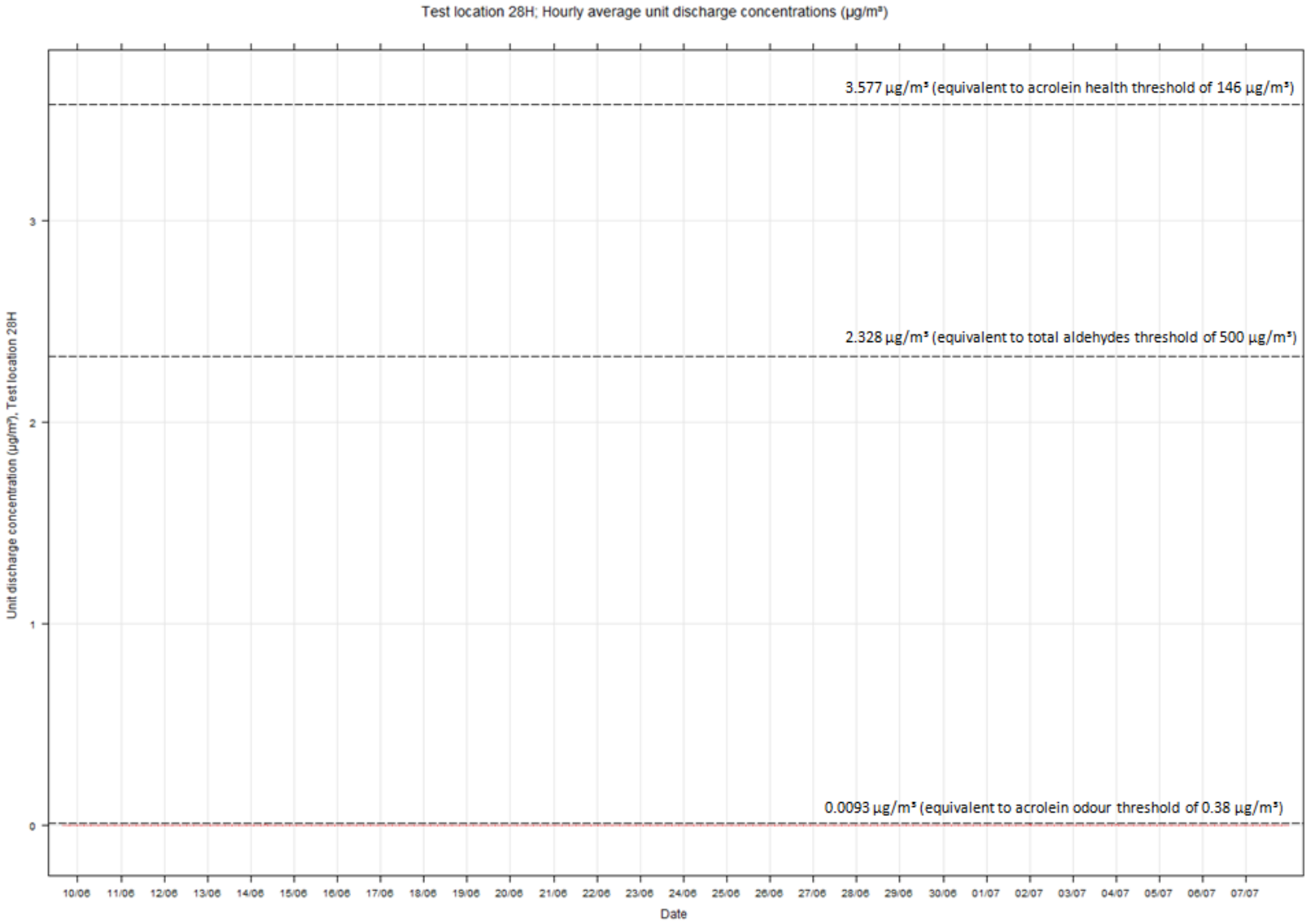

Test location 20; Hourly average unit discharge concentrations (µg/m³)
3.577 µg/m³ (equivalent to acrolein health threshold of 146 µg/m³)
2.328 µg/m³ (equivalent to total aldehydes threshold of 500 µg/m³)
0.0093 µg/m³ (equivalent to acrolein odour threshold of 0.38 µg/m³)
Unit discharge concentration (µg/m³), Test location 20
0
1
2
3
10/06 11/06 12/06 13/06 14/06 15/06 16/06 17/06 18/06 19/06 20/06 21/06 22/06 23/06 24/06 25/06 26/06 27/06 28/06 29/06 30/06 01/07 02/07 03/07 04/07 05/07 06/07 07/07
Date

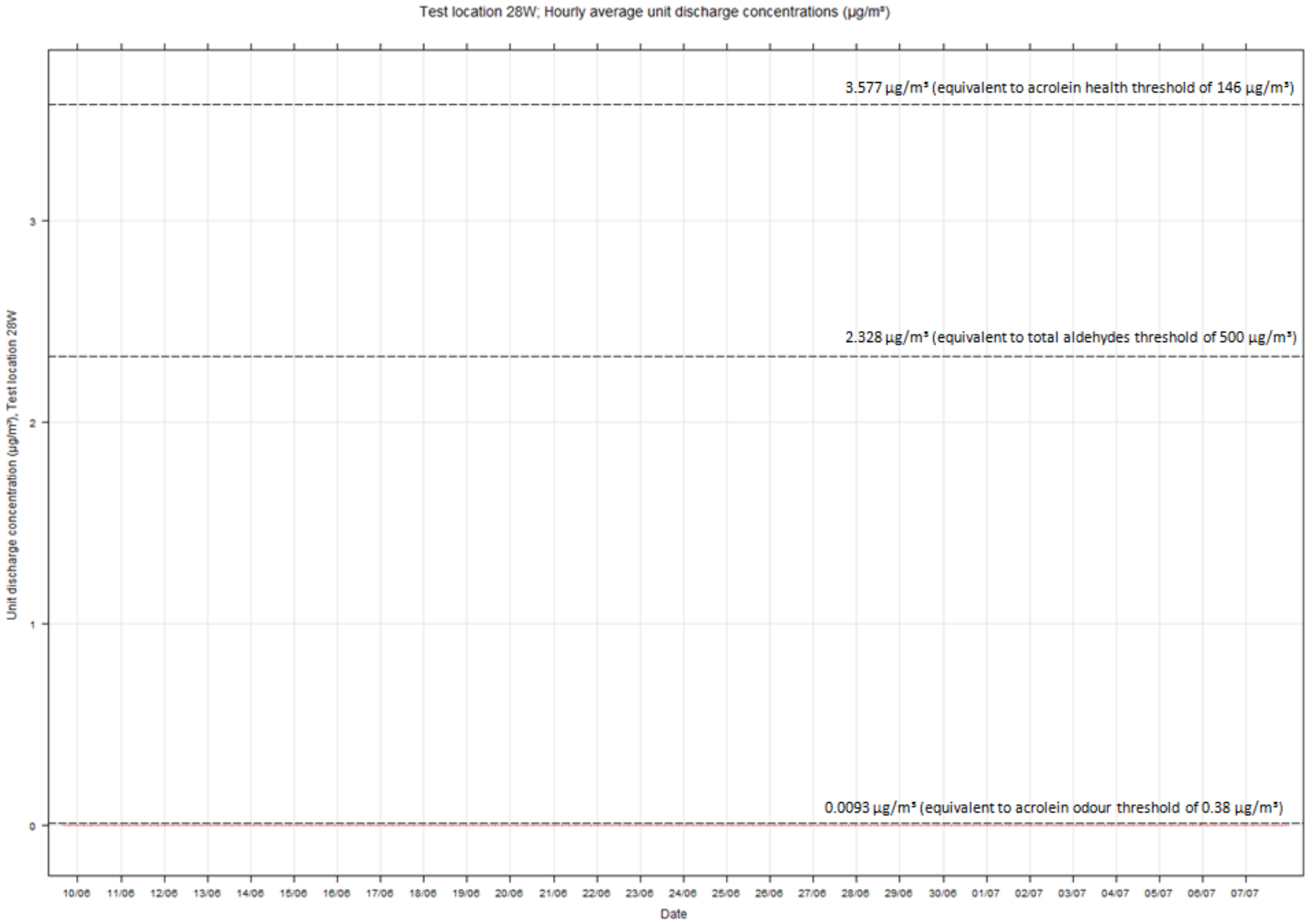

Test location 23; Hourly average unit discharge concentrations (µg/m³)
3.577 µg/m³ (equivalent to acrolein health threshold of 146 µg/m³)
2.328 µg/m³ (equivalent to total aldehydes threshold of 500 µg/m³)
0.0093 µg/m³ (equivalent to acrolein odour threshold of 0.38 µg/m³)
Unit discharge concentration (µg/m³), Test location 23
0
1
2
3
10/06 11/06 12/06 13/06 14/06 15/06 16/06 17/06 18/06 19/06 20/06 21/06 22/06 23/06 24/06 25/06 26/06 27/06 28/06 29/06 30/06 01/07 02/07 03/07 04/07 05/07 06/07 07/07
Date

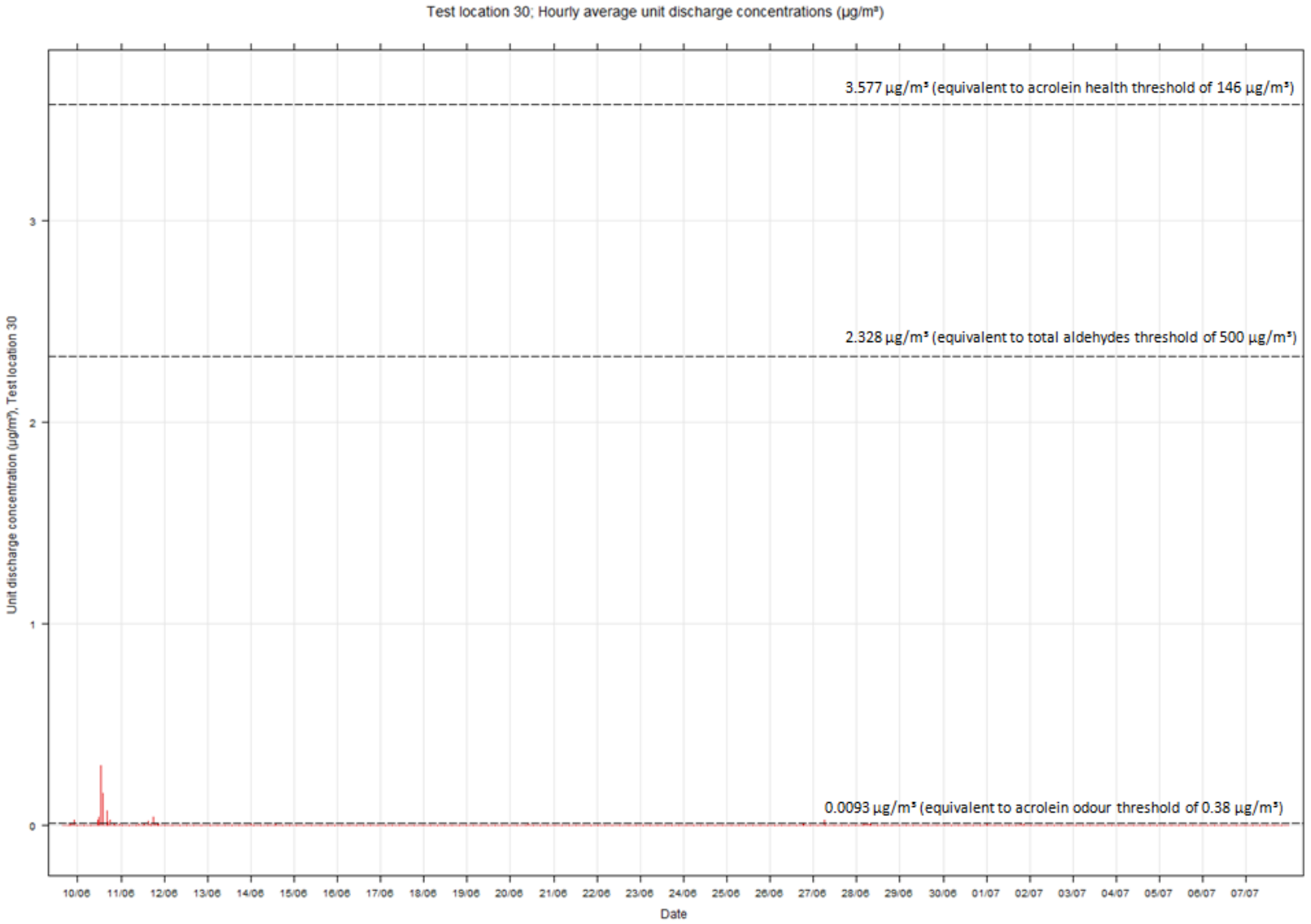

Test location 24; Hourly average unit discharge concentrations (µg/m³)
3.577 µg/m³ (equivalent to acrolein health threshold of 146 µg/m³)
2.328 µg/m³ (equivalent to total aldehydes threshold of 500 µg/m³)
0.0093 µg/m³ (equivalent to acrolein odour threshold of 0.38 µg/m³)
Unit discharge concentration (µg/m³), Test location 24
0
1
2
3
10/06 11/06 12/06 13/06 14/06 15/06 16/06 17/06 18/06 19/06 20/06 21/06 22/06 23/06 24/06 25/06 26/06 27/06 28/06 29/06 30/06 01/07 02/07 03/07 04/07 05/07 06/07 07/07
Date

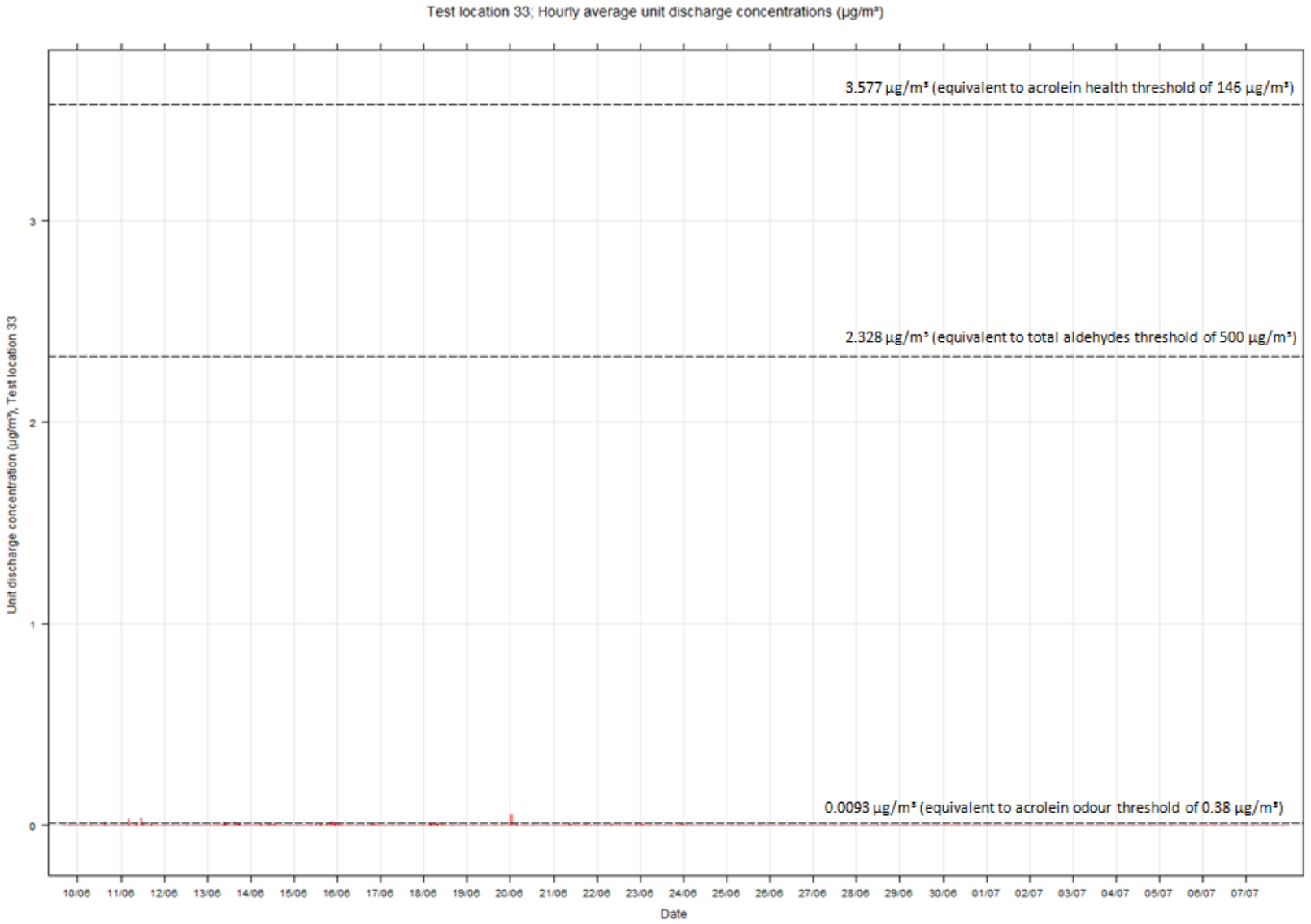

Test location 25; Hourly average unit discharge concentrations (µg/m³)
3.577 µg/m³ (equivalent to acrolein health threshold of 146 µg/m³)
2.328 µg/m³ (equivalent to total aldehydes threshold of 500 µg/m³)
0.0093 µg/m³ (equivalent to acrolein odour threshold of 0.38 µg/m³)
Unit discharge concentration (µg/m³), Test location 25
0
1
2
3
10/06
11/06
12/06
13/06
14/06
15/06
16/06
17/06
18/06
19/06
20/06
21/06
22/06
23/06
24/06
25/06
26/06
27/06
28/06
29/06
30/06
01/07
02/07
03/07
04/07
05/07
06/07
07/07
Date

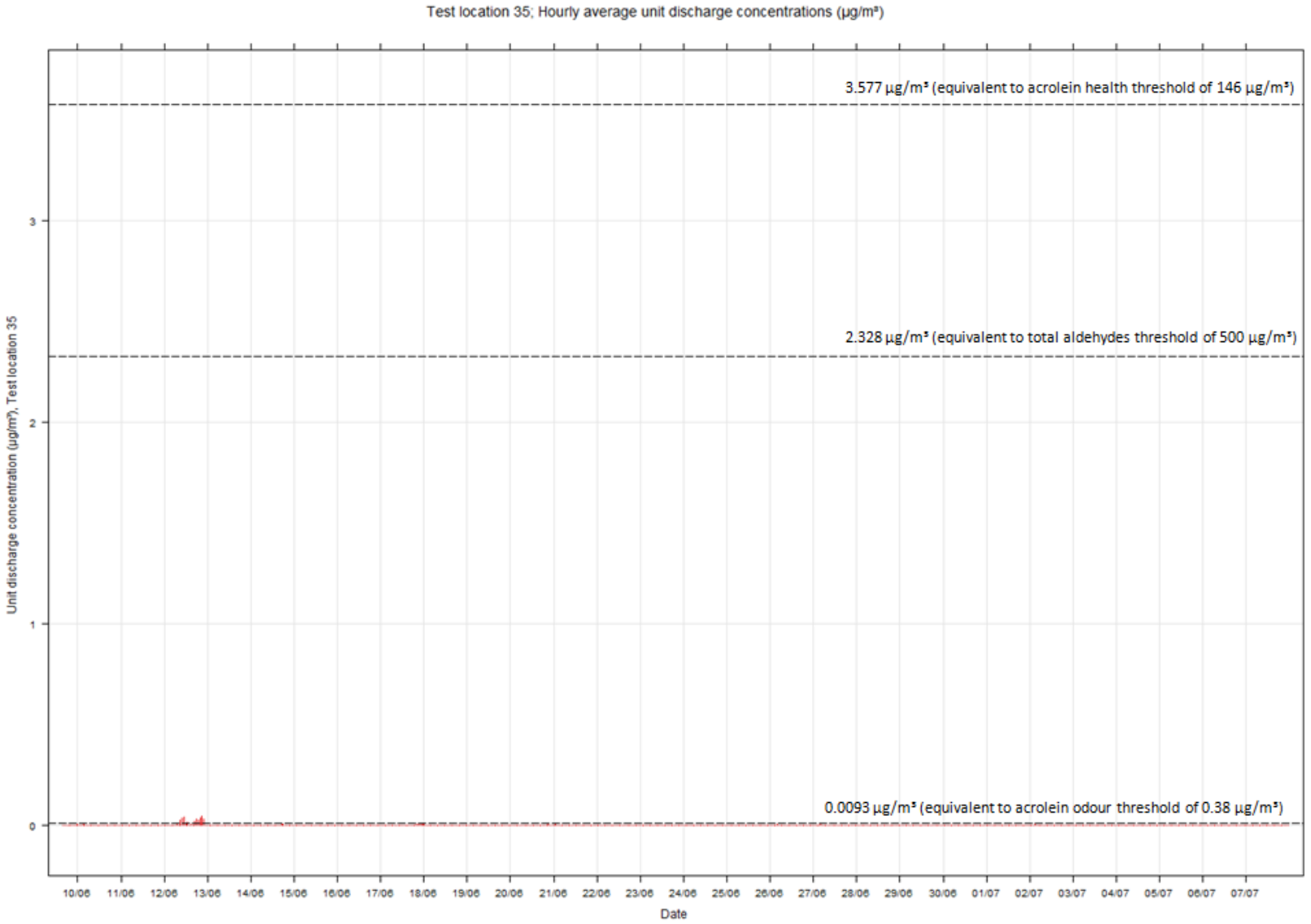

Test location 27; Hourly average unit discharge concentrations (µg/m³)
3.577 µg/m³ (equivalent to acrolein health threshold of 146 µg/m³)
2.328 µg/m³ (equivalent to total aldehydes threshold of 500 µg/m³)
0.0093 µg/m³ (equivalent to acrolein odour threshold of 0.38 µg/m³)
Unit discharge concentration (µg/m³), Test location 27
0
1
2
3
10/06 11/06 12/06 13/06 14/06 15/06 16/06 17/06 18/06 19/06 20/06 21/06 22/06 23/06 24/06 25/06 26/06 27/06 28/06 29/06 30/06 01/07 02/07 03/07 04/07 05/07 06/07 07/07
Date

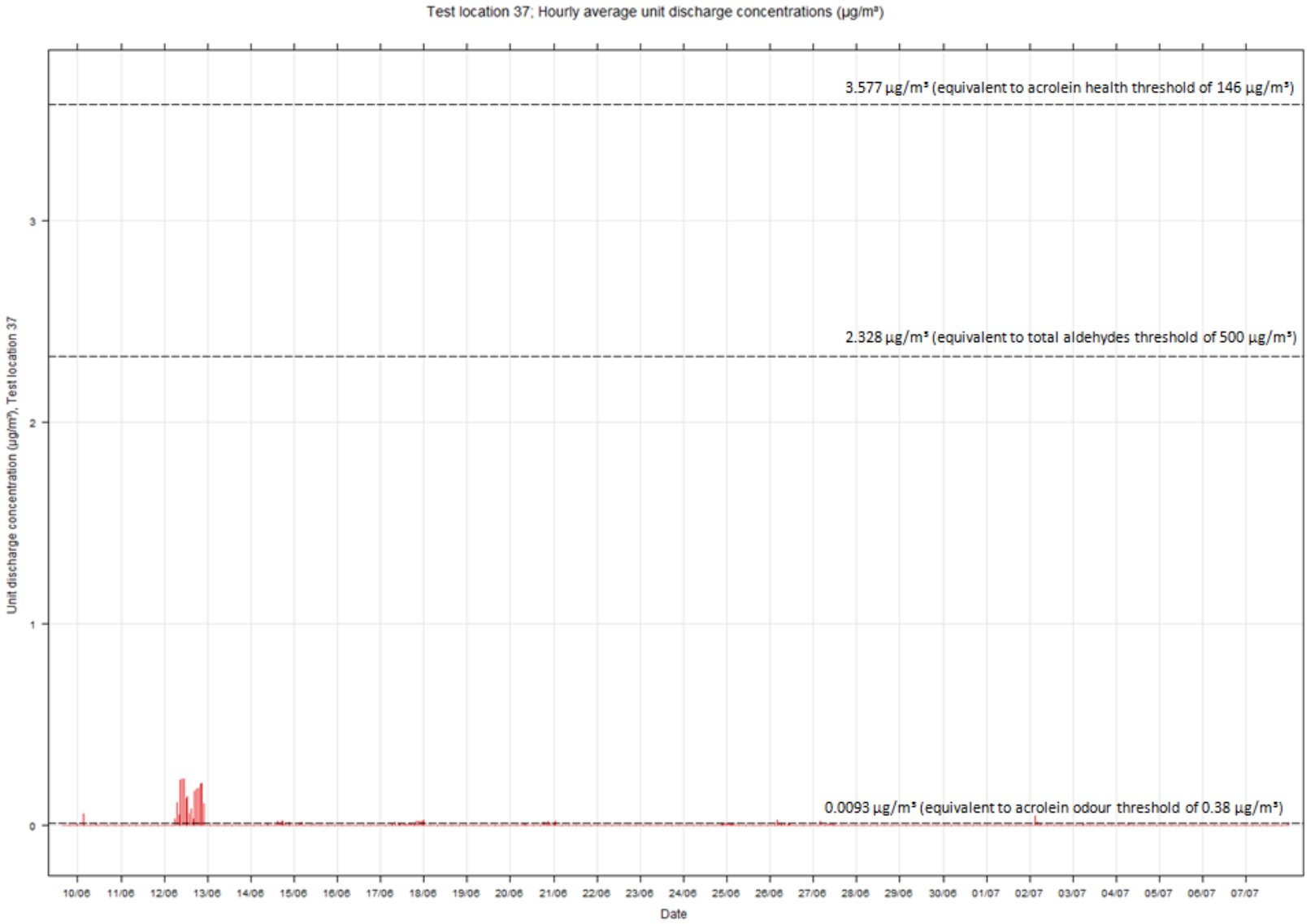

Test location 28H; Hourly average unit discharge concentrations (µg/m³)
3.577 µg/m³ (equivalent to acrolein health threshold of 146 µg/m³)
2.328 µg/m³ (equivalent to total aldehydes threshold of 500 µg/m³)
0.0093 µg/m³ (equivalent to acrolein odour threshold of 0.38 µg/m³)
Unit discharge concentration (µg/m³), Test location 28H
0
1
2
3
10/06 11/06 12/06 13/06 14/06 15/06 16/06 17/06 18/06 19/06 20/06 21/06 22/06 23/06 24/06 25/06 26/06 27/06 28/06 29/06 30/06 01/07 02/07 03/07 04/07 05/07 06/07 07/07
Date

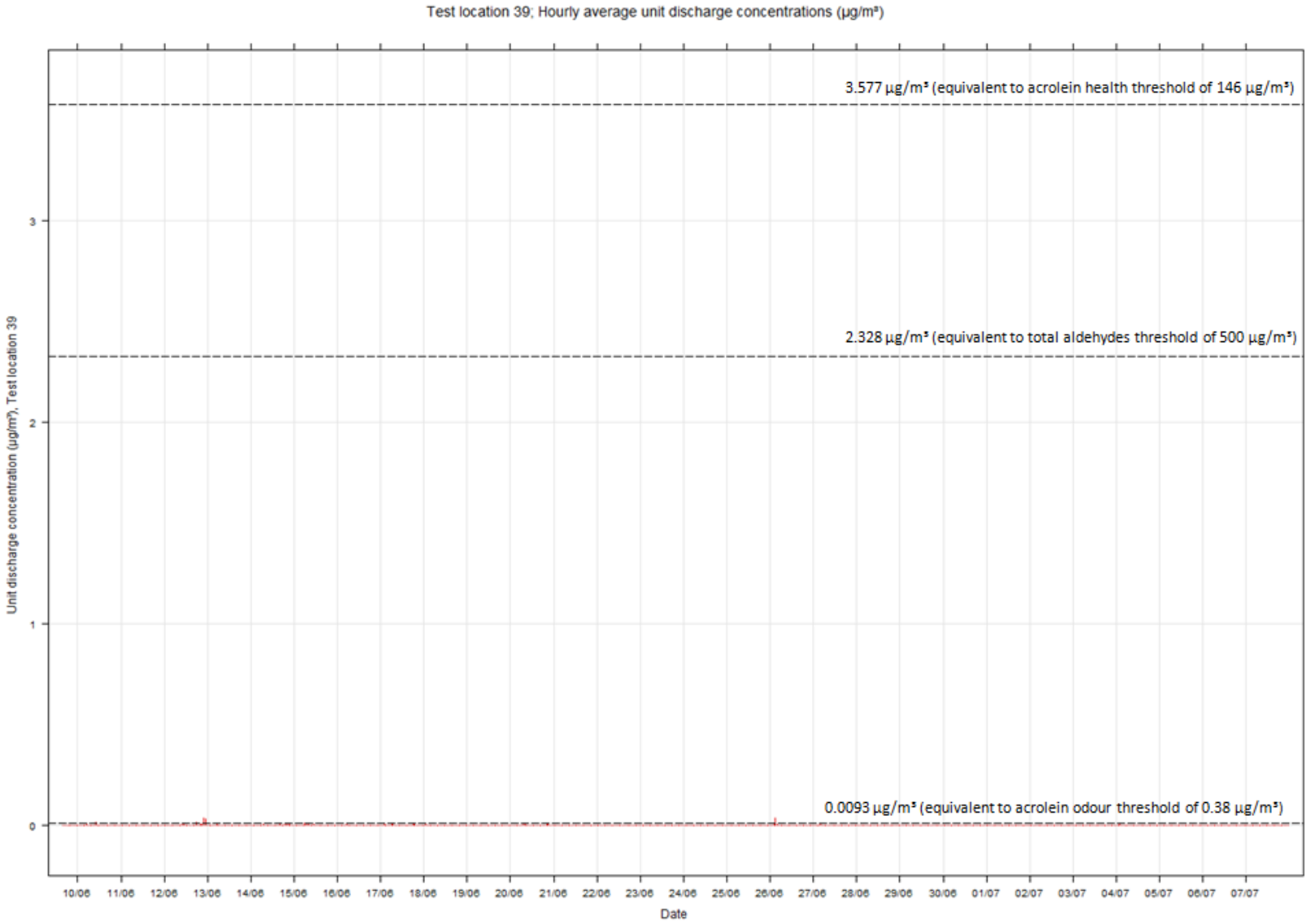

Test location 28W; Hourly average unit discharge concentrations (µg/m³)
3.577 µg/m³ (equivalent to acrolein health threshold of 146 µg/m³)
2.328 µg/m³ (equivalent to total aldehydes threshold of 500 µg/m³)
0.0093 µg/m³ (equivalent to acrolein odour threshold of 0.38 µg/m³)
Unit discharge concentration (µg/m³), Test location 28W
0
1
2
3
10/06 11/06 12/06 13/06 14/06 15/06 16/06 17/06 18/06 19/06 20/06 21/06 22/06 23/06 24/06 25/06 26/06 27/06 28/06 29/06 30/06 01/07 02/07 03/07 04/07 05/07 06/07 07/07
Date

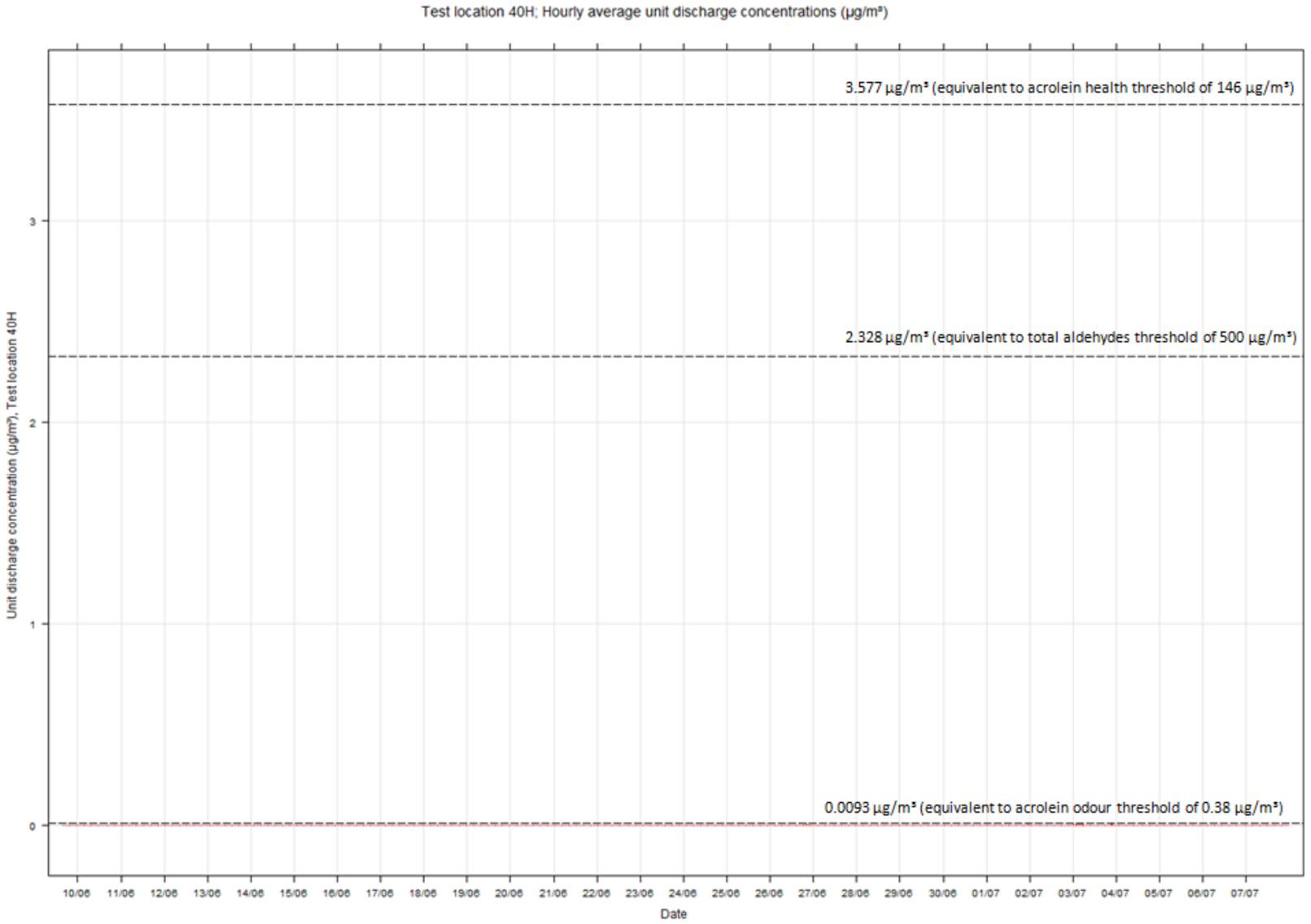

Test location 30; Hourly average unit discharge concentrations (µg/m³)
3.577 µg/m³ (equivalent to acrolein health threshold of 146 µg/m³)
2.328 µg/m³ (equivalent to total aldehydes threshold of 500 µg/m³)
0.0093 µg/m³ (equivalent to acrolein odour threshold of 0.38 µg/m³)
Unit discharge concentration (µg/m³), Test location 30
0
1
2
3
10/06 11/06 12/06 13/06 14/06 15/06 16/06 17/06 18/06 19/06 20/06 21/06 22/06 23/06 24/06 25/06 26/06 27/06 28/06 29/06 30/06 01/07 02/07 03/07 04/07 05/07 06/07 07/07
Date

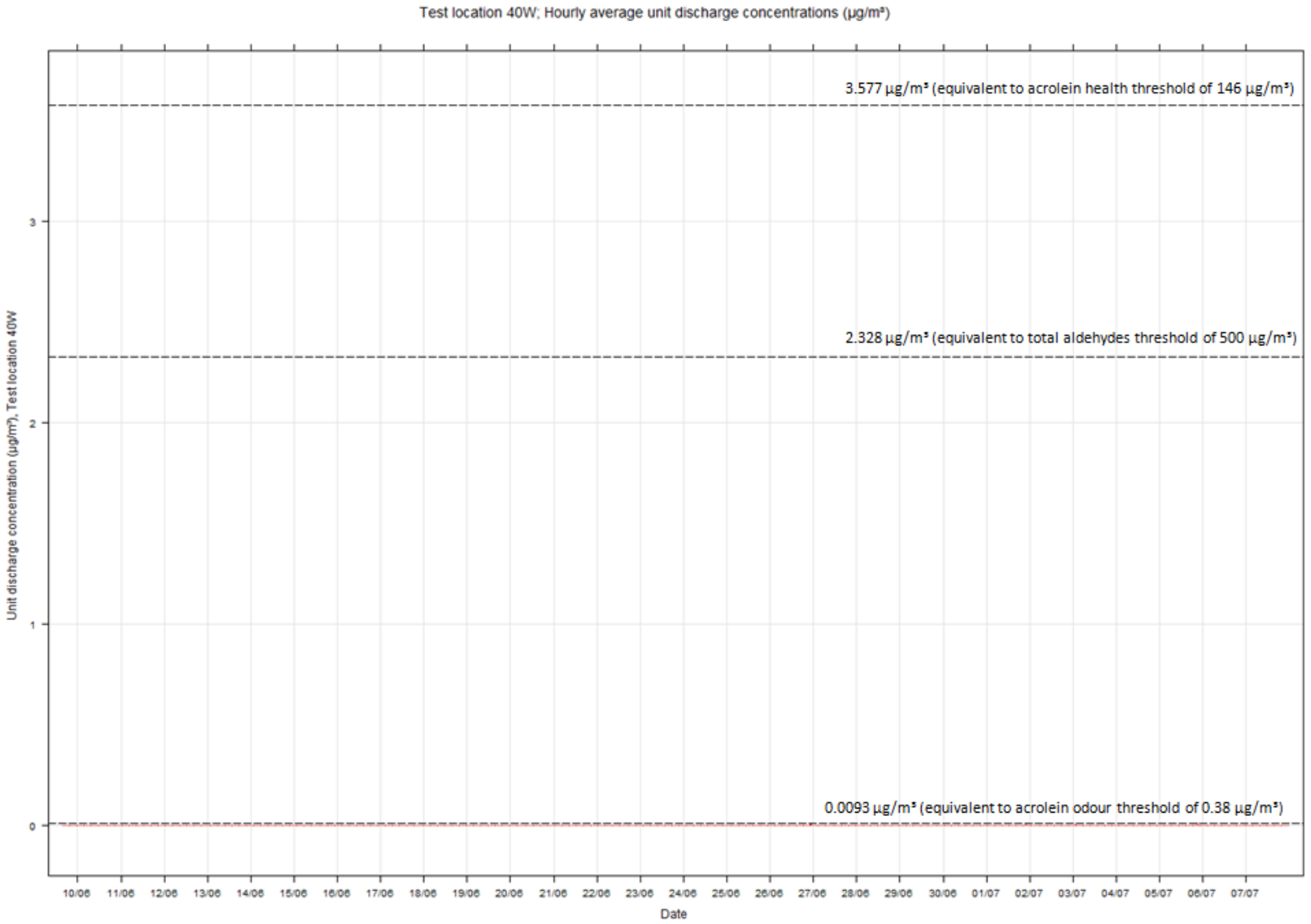

Test location 33; Hourly average unit discharge concentrations (µg/m³)
3.577 µg/m³ (equivalent to acrolein health threshold of 146 µg/m³)
2.328 µg/m³ (equivalent to total aldehydes threshold of 500 µg/m³)
0.0093 µg/m³ (equivalent to acrolein odour threshold of 0.38 µg/m³)
Unit discharge concentration (µg/m³), Test location 33
0
1
2
3
10/06 11/06 12/06 13/06 14/06 15/06 16/06 17/06 18/06 19/06 20/06 21/06 22/06 23/06 24/06 25/06 26/06 27/06 28/06 29/06 30/06 01/07 02/07 03/07 04/07 05/07 06/07 07/07
Date


Test location 35; Hourly average unit discharge concentrations (µg/m³)
3.577 µg/m³ (equivalent to acrolein health threshold of 146 µg/m³)
2.328 µg/m³ (equivalent to total aldehydes threshold of 500 µg/m³)
0.0093 µg/m³ (equivalent to acrolein odour threshold of 0.38 µg/m³)
Unit discharge concentration (µg/m³), Test location 35
0
1
2
3
10/06 11/06 12/06 13/06 14/06 15/06 16/06 17/06 18/06 19/06 20/06 21/06 22/06 23/06 24/06 25/06 26/06 27/06 28/06 29/06 30/06 01/07 02/07 03/07 04/07 05/07 06/07 07/07
Date


Test location 37; Hourly average unit discharge concentrations (µg/m³)
3.577 µg/m³ (equivalent to acrolein health threshold of 146 µg/m³)
2.328 µg/m³ (equivalent to total aldehydes threshold of 500 µg/m³)
0.0093 µg/m³ (equivalent to acrolein odour threshold of 0.38 µg/m³)
Unit discharge concentration (µg/m³), Test location 37
0
1
2
3
10/06 11/06 12/06 13/06 14/06 15/06 16/06 17/06 18/06 19/06 20/06 21/06 22/06 23/06 24/06 25/06 26/06 27/06 28/06 29/06 30/06 01/07 02/07 03/07 04/07 05/07 06/07 07/07
Date


Test location 39; Hourly average unit discharge concentrations (µg/m³)
3.577 µg/m³ (equivalent to acrolein health threshold of 146 µg/m³)
2.328 µg/m³ (equivalent to total aldehydes threshold of 500 µg/m³)
0.0093 µg/m³ (equivalent to acrolein odour threshold of 0.38 µg/m³)
Unit discharge concentration (µg/m³), Test location 39
0
1
2
3
10/06 11/06 12/06 13/06 14/06 15/06 16/06 17/06 18/06 19/06 20/06 21/06 22/06 23/06 24/06 25/06 26/06 27/06 28/06 29/06 30/06 01/07 02/07 03/07 04/07 05/07 06/07 07/07
Date


Test location 40H; Hourly average unit discharge concentrations (µg/m³)
3.577 µg/m³ (equivalent to acrolein health threshold of 146 µg/m³)
2.328 µg/m³ (equivalent to total aldehydes threshold of 500 µg/m³)
0.0093 µg/m³ (equivalent to acrolein odour threshold of 0.38 µg/m³)
Unit discharge concentration (µg/m³), Test location 40H
0
1
2
3
10/06 11/06 12/06 13/06 14/06 15/06 16/06 17/06 18/06 19/06 20/06 21/06 22/06 23/06 24/06 25/06 26/06 27/06 28/06 29/06 30/06 01/07 02/07 03/07 04/07 05/07 06/07 07/07
Date


Test location 40W; Hourly average unit discharge concentrations (µg/m³)
3.577 µg/m³ (equivalent to acrolein health threshold of 146 µg/m³)
2.328 µg/m³ (equivalent to total aldehydes threshold of 500 µg/m³)
0.0093 µg/m³ (equivalent to acrolein odour threshold of 0.38 µg/m³)
Unit discharge concentration (µg/m³), Test location 40W
0
1
2
3
10/06 11/06 12/06 13/06 14/06 15/06 16/06 17/06 18/06 19/06 20/06 21/06 22/06 23/06 24/06 25/06 26/06 27/06 28/06 29/06 30/06 01/07 02/07 03/07 04/07 05/07 06/07 07/07
Date

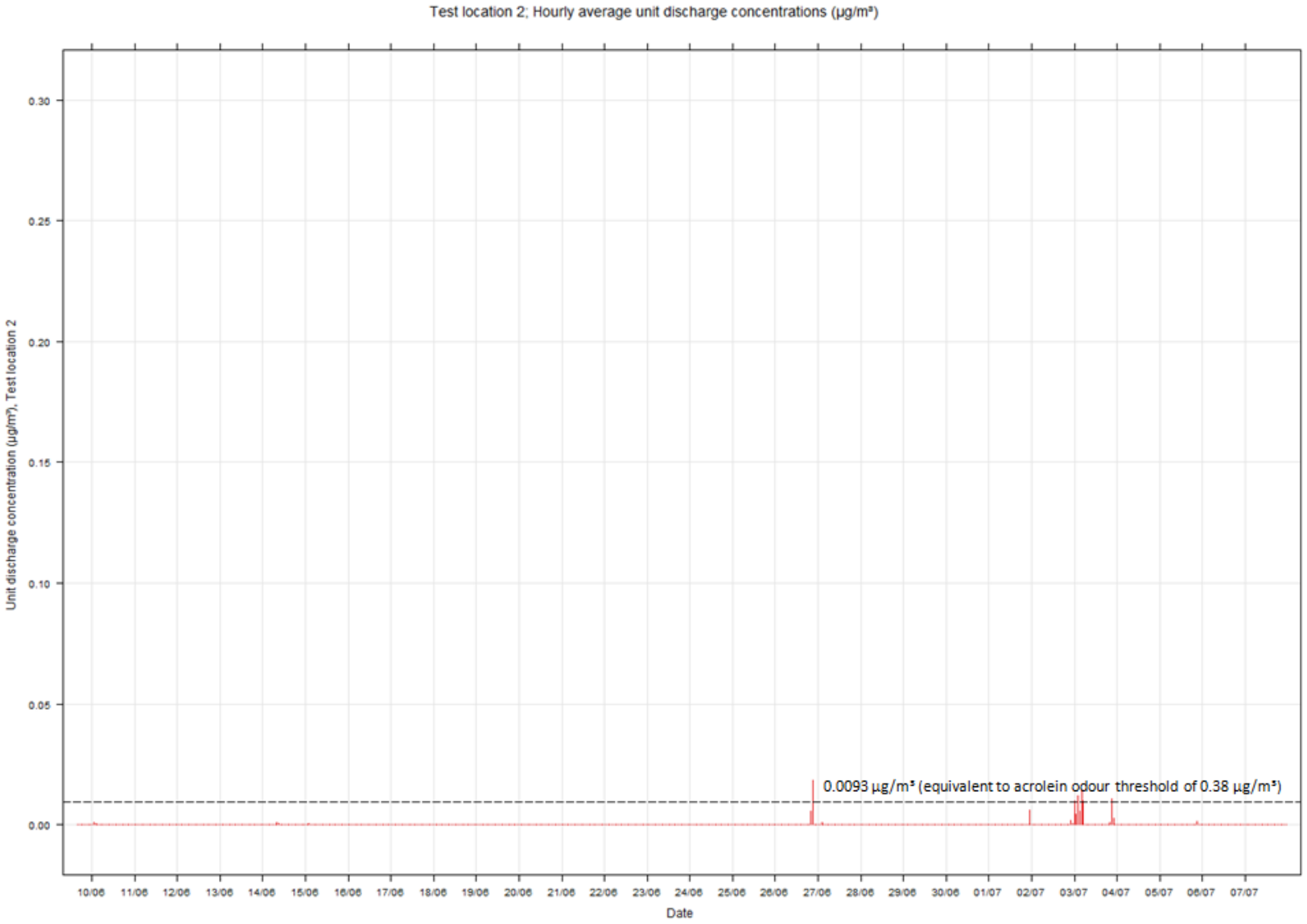
Sonae GLO: Further Modelling

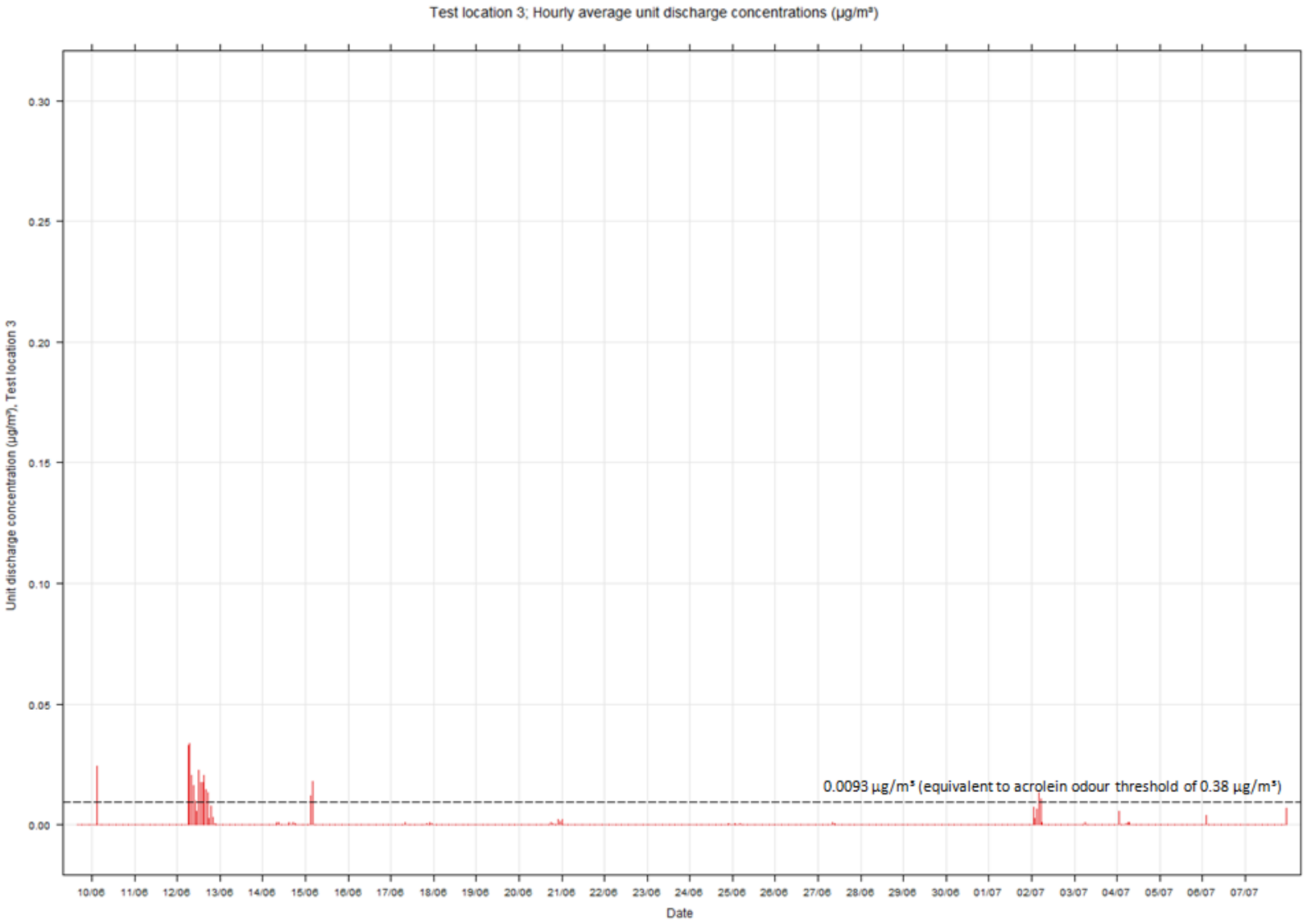
4a. Histograms of modelled hourly concentrations for the 20 Test Claimants

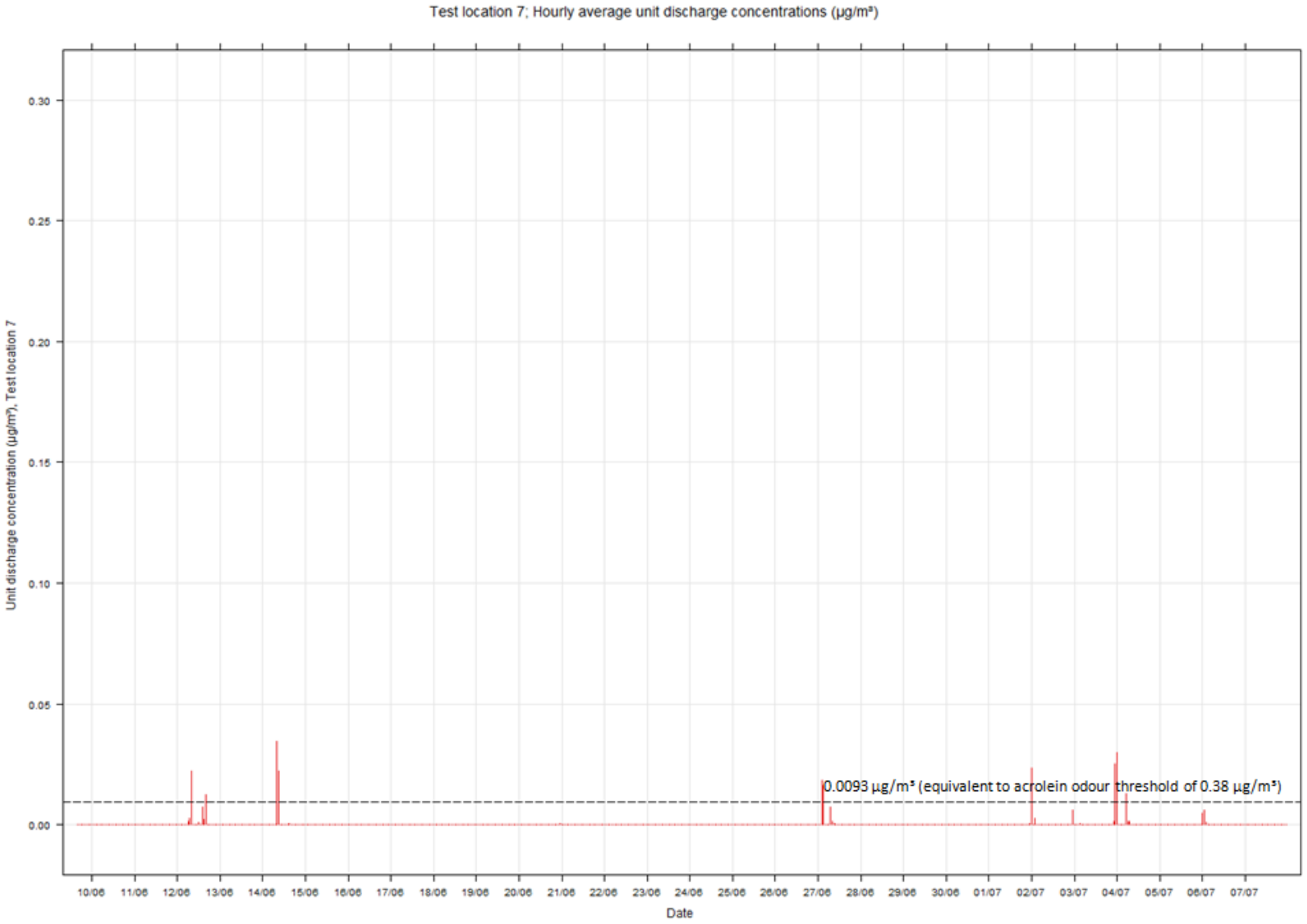
ii) *Unit discharge concentrations showing only the equivalent threshold for the acrolein odour threshold*

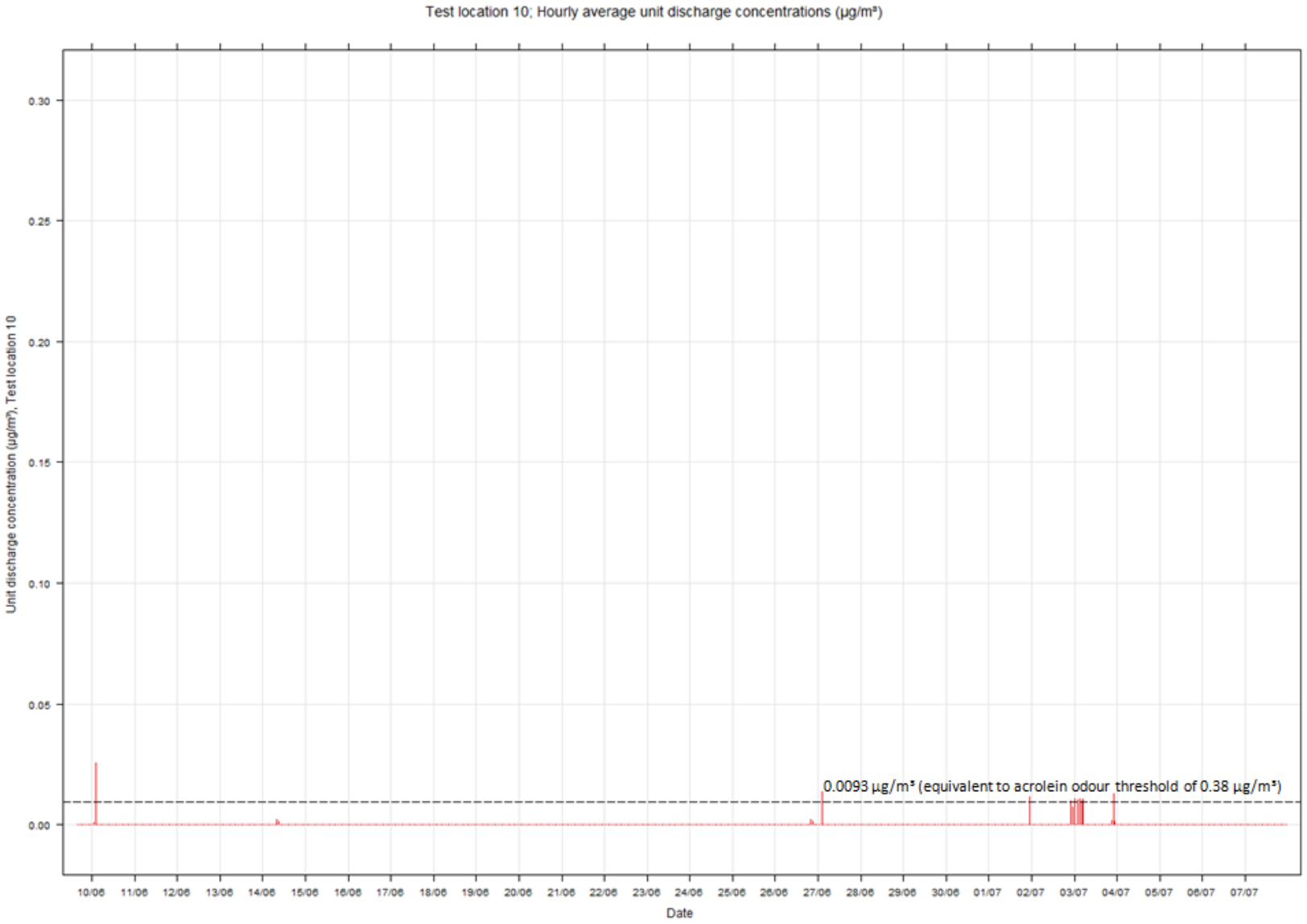
This document presents time series of hourly average concentrations for a 'unit discharge' at the locations of the 20 Test Claimants.

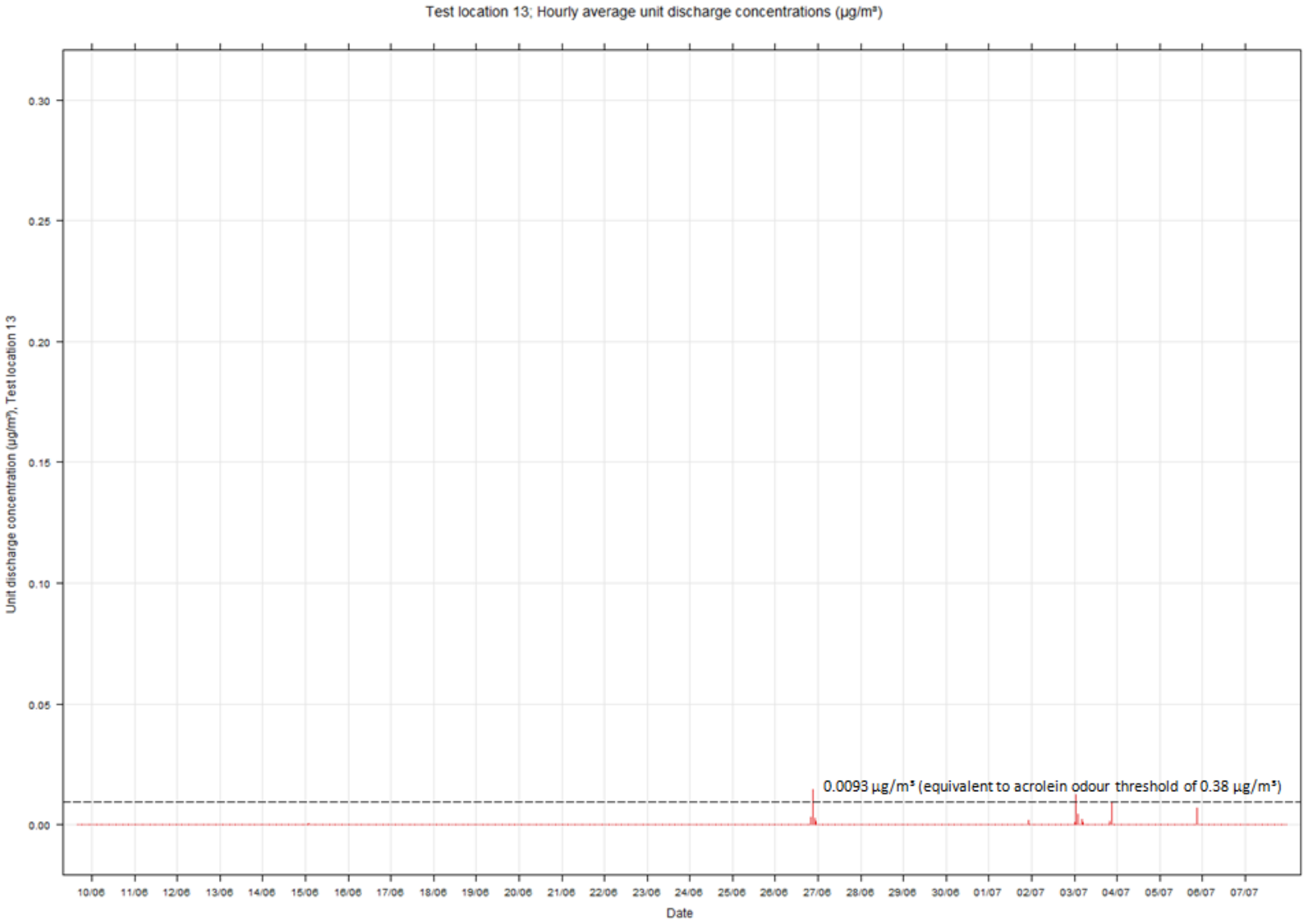
Since the maximum modelled hourly unit discharge concentration across the Test Claimant locations is 0.29 µg/m³, the 23 histograms shown in Document 4 are presented again in this document with a larger scale on the 'y' axis (maximum concentration is set to 0.3 µg/m³). Consequently only the equivalent threshold for the acrolein odour threshold of 0.38 µg/m³ (equivalent to a unit discharge concentration of 0.0093 µg/m³) can be shown on these histograms.

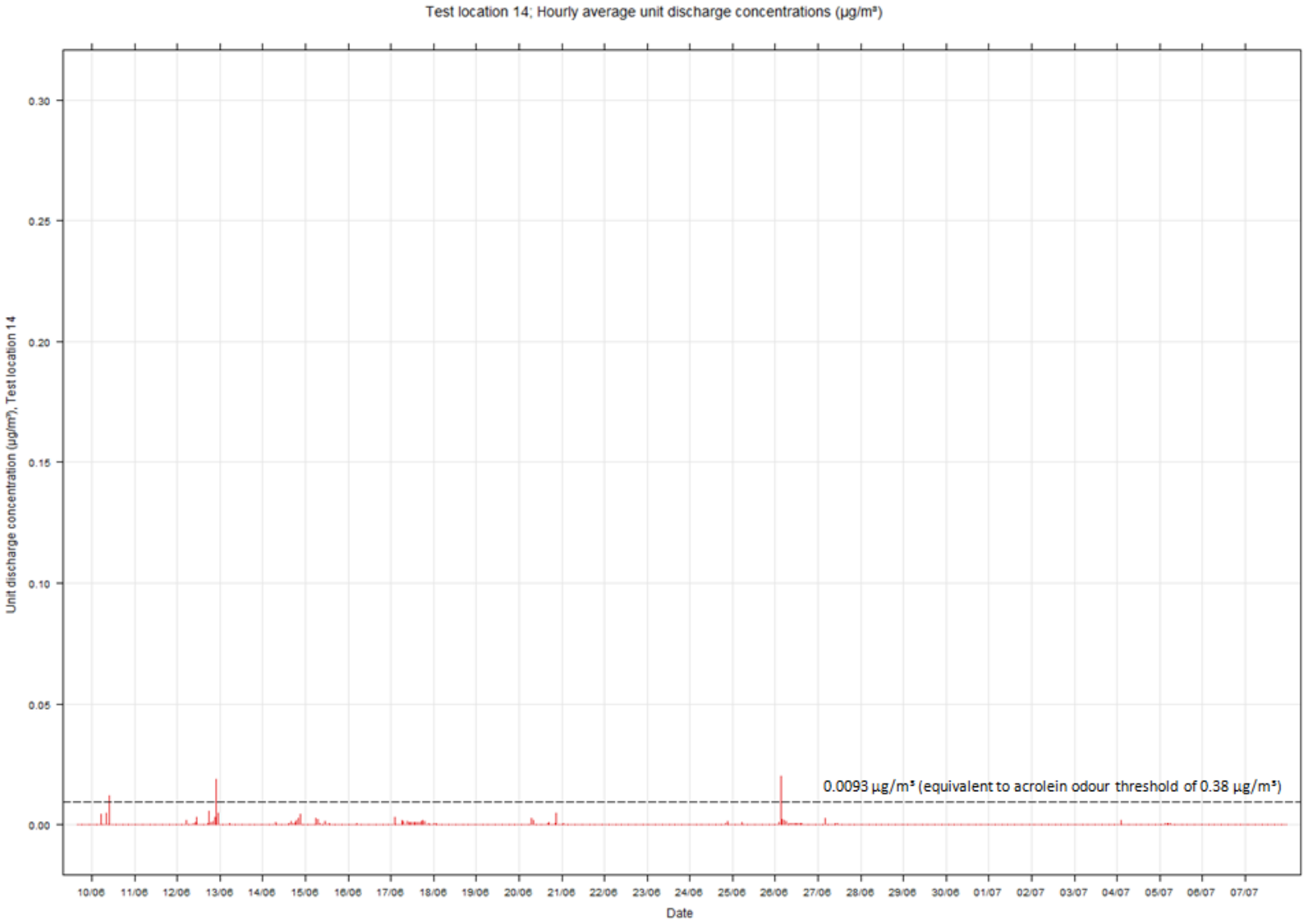

Test location 2; Hourly average unit discharge concentrations (µg/m³)
Unit discharge concentration (µg/m³), Test location 2
0.30
0.25
0.20
0.15
0.10
0.05
0.00
0.0093 µg/m³ (equivalent to acrolein odour threshold of 0.38 µg/m³)
10/06
11/06
12/06
13/06
14/06
15/06
16/06
17/06
18/06
19/06
20/06
21/06
22/06
23/06
24/06
25/06
26/06
27/06
28/06
29/06
30/06
01/07
02/07
03/07
04/07
05/07
06/07
07/07
Date

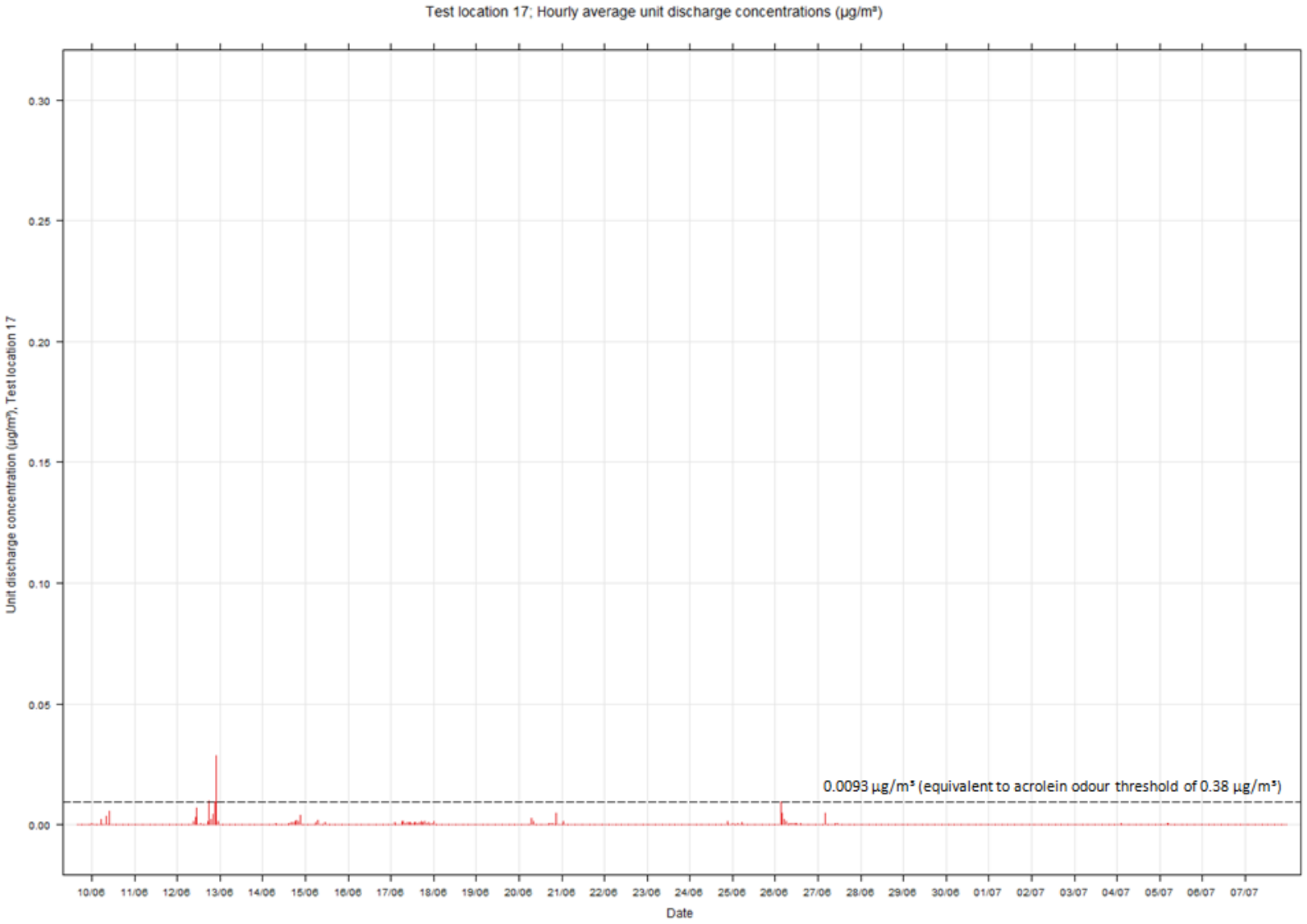

Test location 3; Hourly average unit discharge concentrations (µg/m³)
Unit discharge concentration (µg/m³), Test location 3
0.30
0.25
0.20
0.15
0.10
0.05
0.00
0.0093 µg/m³ (equivalent to acrolein odour threshold of 0.38 µg/m³)
10/06
11/06
12/06
13/06
14/06
15/06
16/06
17/06
18/06
19/06
20/06
21/06
22/06
23/06
24/06
25/06
26/06
27/06
28/06
29/06
30/06
01/07
02/07
03/07
04/07
05/07
06/07
07/07
Date

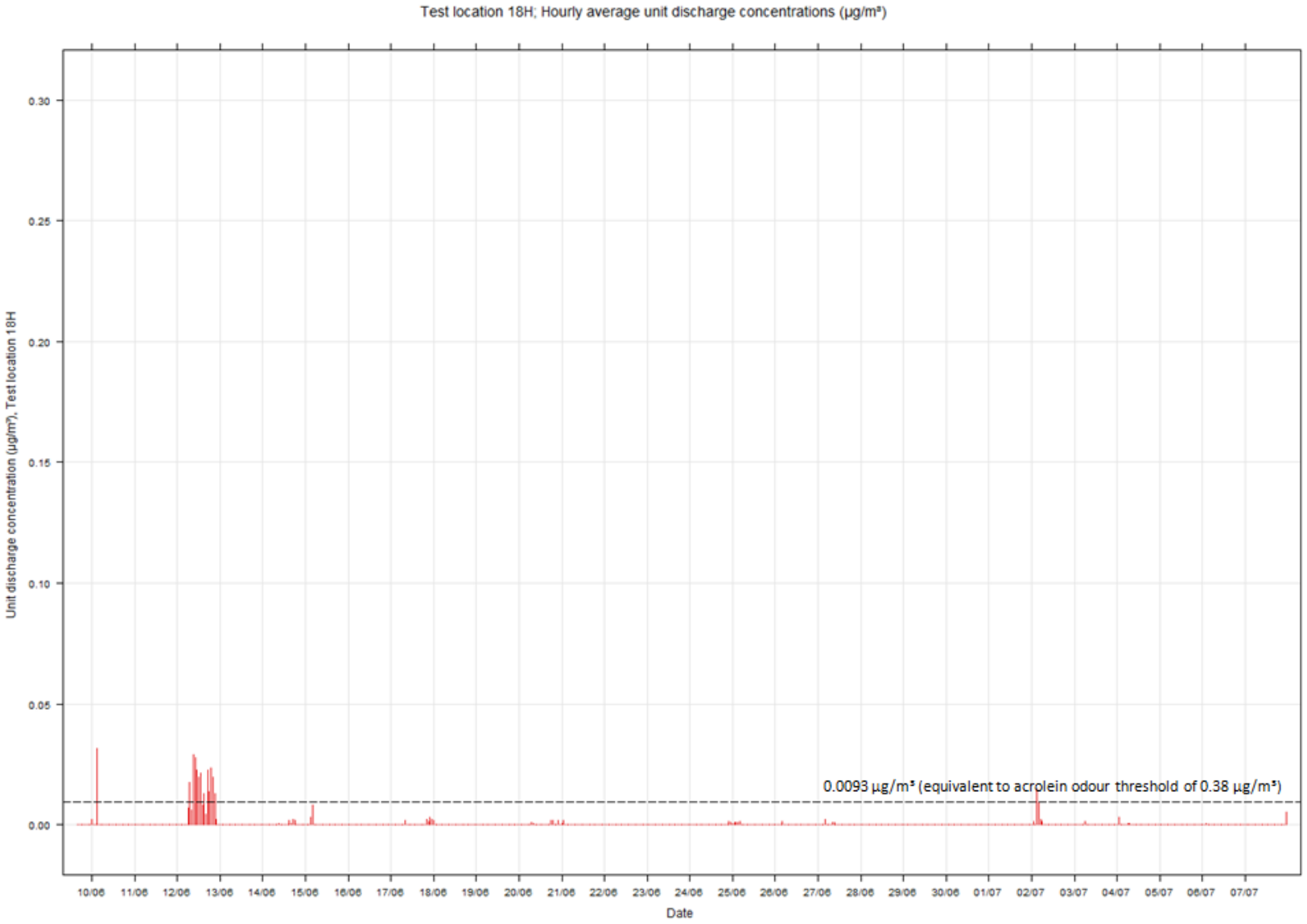

Test location 7; Hourly average unit discharge concentrations (µg/m³)
Unit discharge concentration (µg/m³), Test location 7
0.30
0.25
0.20
0.15
0.10
0.05
0.00
0.0093 µg/m³ (equivalent to acrolein odour threshold of 0.38 µg/m³)
10/06
11/06
12/06
13/06
14/06
15/06
16/06
17/06
18/06
19/06
20/06
21/06
22/06
23/06
24/06
25/06
26/06
27/06
28/06
29/06
30/06
01/07
02/07
03/07
04/07
05/07
06/07
07/07
Date

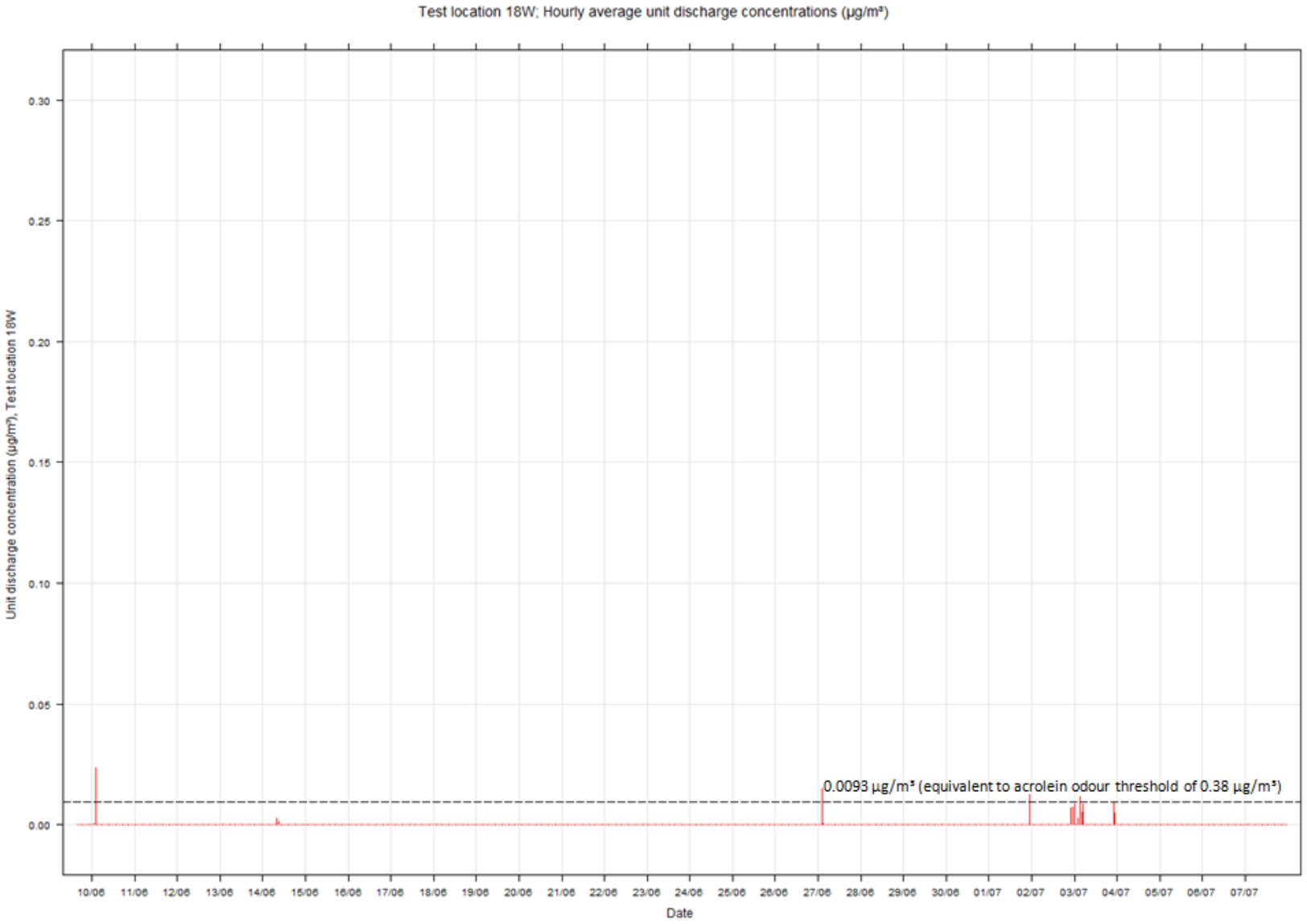

Test location 10; Hourly average unit discharge concentrations (µg/m³)
Unit discharge concentration (µg/m³), Test location 10
0.30
0.25
0.20
0.15
0.10
0.05
0.00
0.0093 µg/m³ (equivalent to acrolein odour threshold of 0.38 µg/m³)
10/06
11/06
12/06
13/06
14/06
15/06
16/06
17/06
18/06
19/06
20/06
21/06
22/06
23/06
24/06
25/06
26/06
27/06
28/06
29/06
30/06
01/07
02/07
03/07
04/07
05/07
06/07
07/07
Date

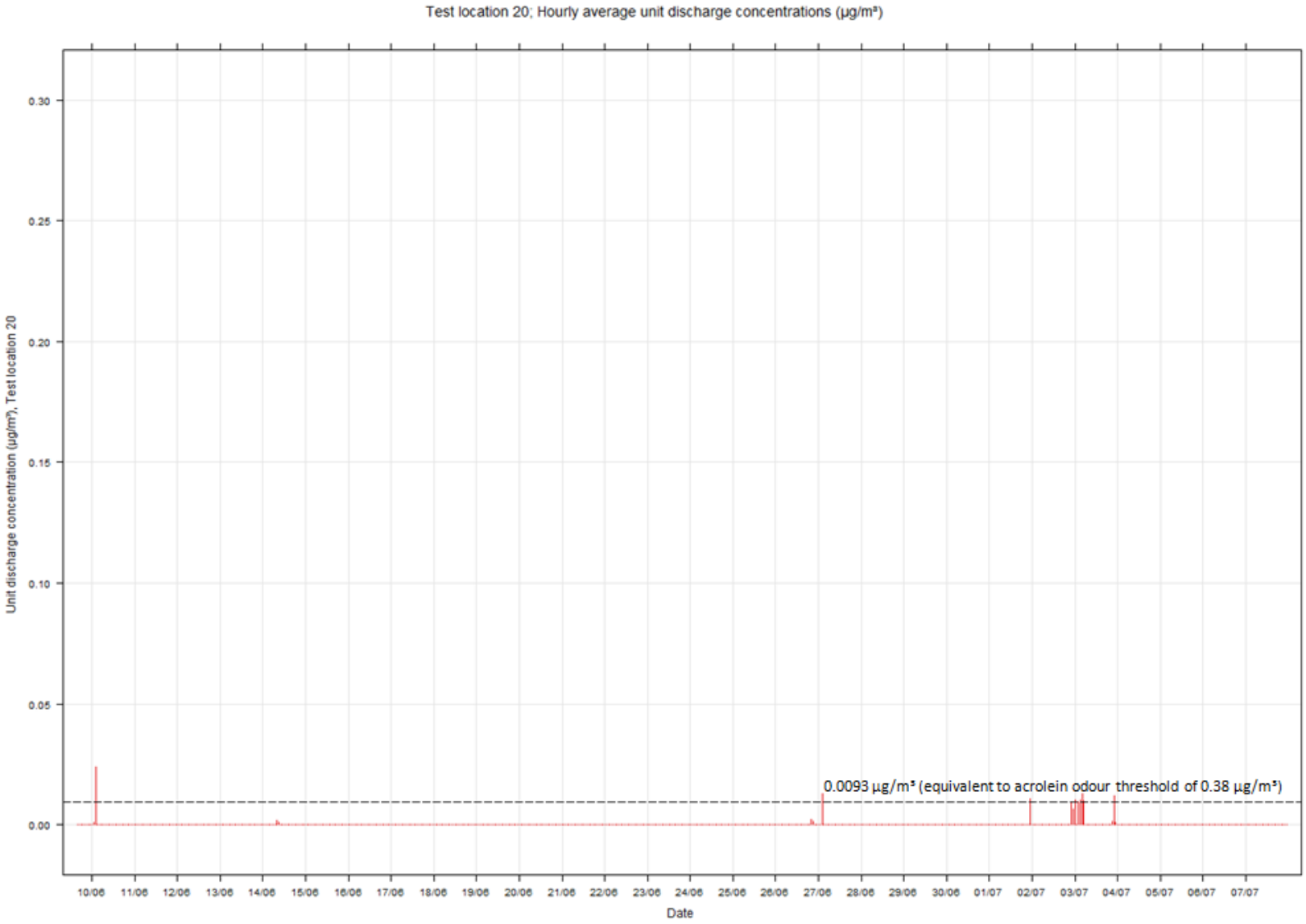

Test location 13; Hourly average unit discharge concentrations (µg/m³)
Unit discharge concentration (µg/m³), Test location 13
0.30
0.25
0.20
0.15
0.10
0.05
0.00
0.0093 µg/m³ (equivalent to acrolein odour threshold of 0.38 µg/m³)
10/06
11/06
12/06
13/06
14/06
15/06
16/06
17/06
18/06
19/06
20/06
21/06
22/06
23/06
24/06
25/06
26/06
27/06
28/06
29/06
30/06
01/07
02/07
03/07
04/07
05/07
06/07
07/07
Date

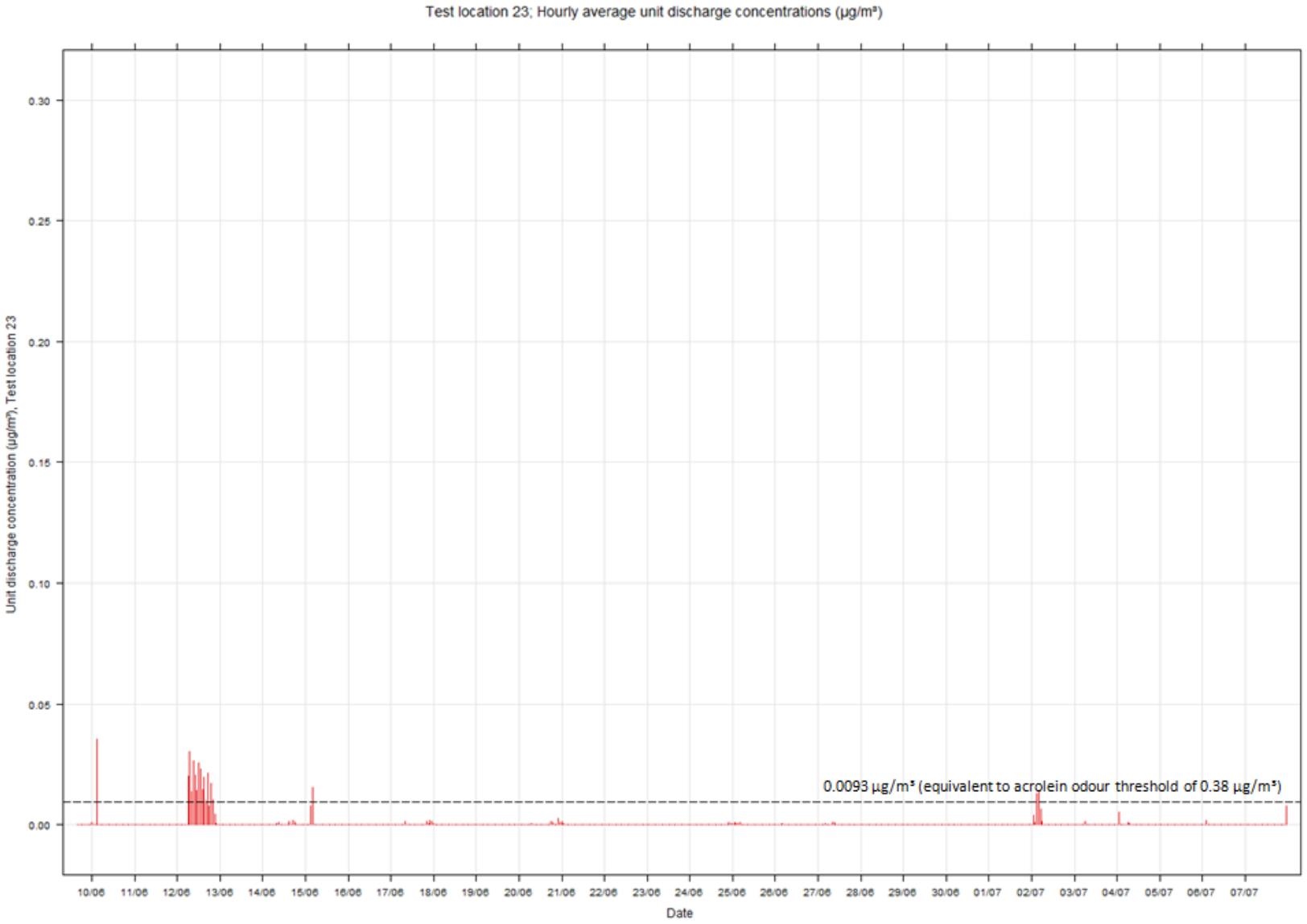

Test location 14; Hourly average unit discharge concentrations (µg/m³)
Unit discharge concentration (µg/m³), Test location 14
0.30
0.25
0.20
0.15
0.10
0.05
0.00
0.0093 µg/m³ (equivalent to acrolein odour threshold of 0.38 µg/m³)
10/06
11/06
12/06
13/06
14/06
15/06
16/06
17/06
18/06
19/06
20/06
21/06
22/06
23/06
24/06
25/06
26/06
27/06
28/06
29/06
30/06
01/07
02/07
03/07
04/07
05/07
06/07
07/07
Date

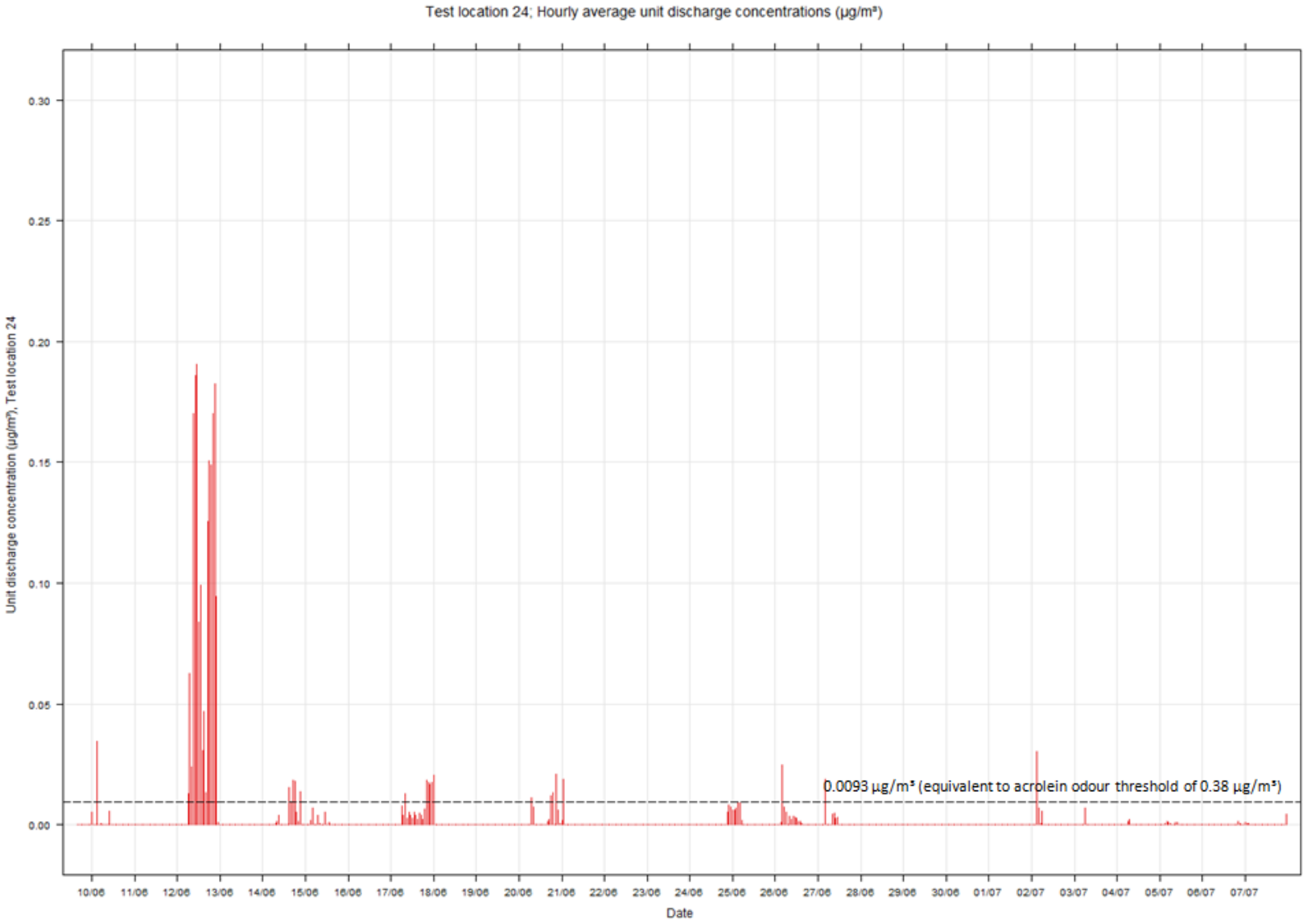

Test location 17; Hourly average unit discharge concentrations (µg/m³)
Unit discharge concentration (µg/m³), Test location 17
0.30
0.25
0.20
0.15
0.10
0.05
0.00
0.0093 µg/m³ (equivalent to acrolein odour threshold of 0.38 µg/m³)
10/06
11/06
12/06
13/06
14/06
15/06
16/06
17/06
18/06
19/06
20/06
21/06
22/06
23/06
24/06
25/06
26/06
27/06
28/06
29/06
30/06
01/07
02/07
03/07
04/07
05/07
06/07
07/07
Date

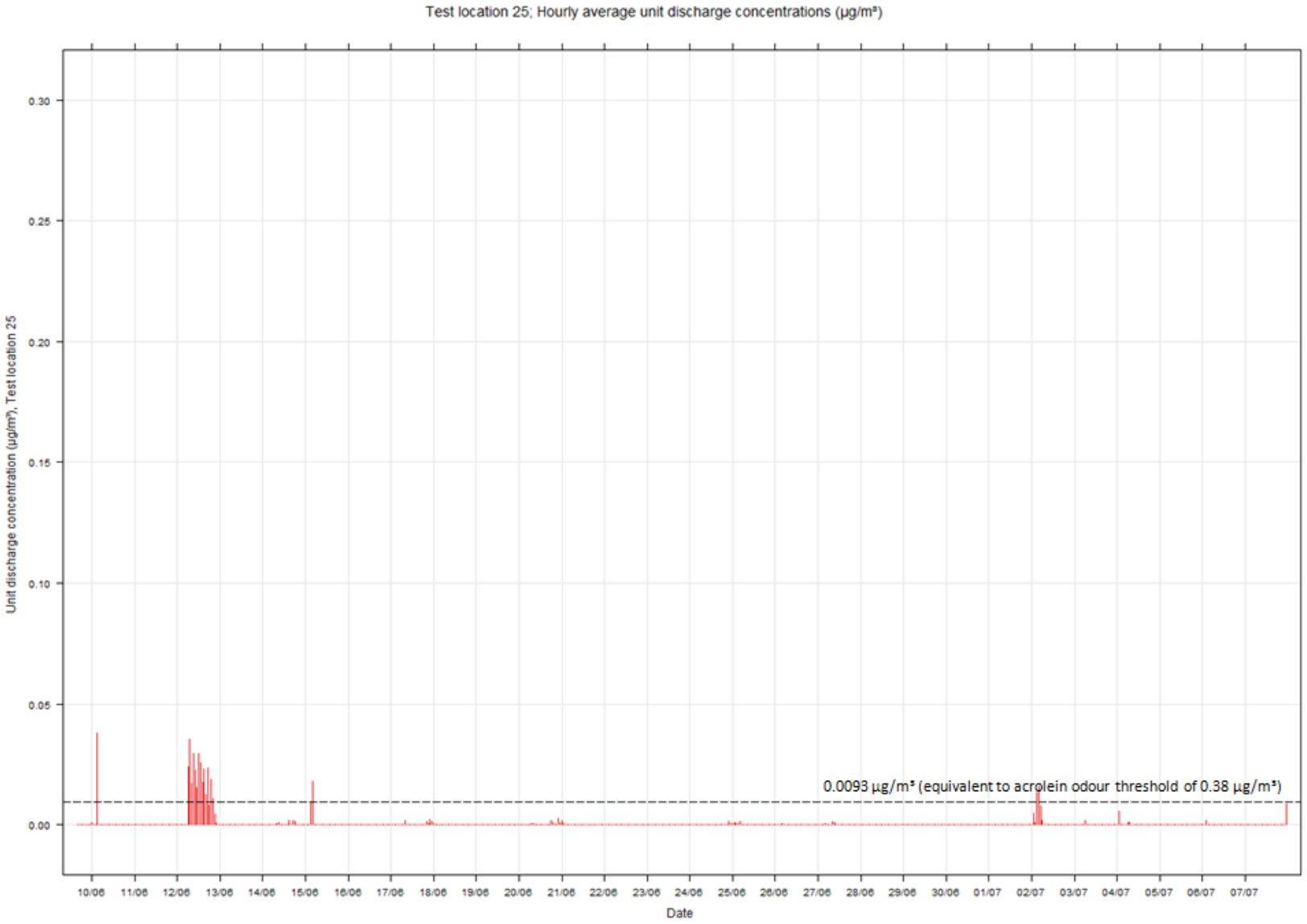

Test location 18H; Hourly average unit discharge concentrations (µg/m³)
Unit discharge concentration (µg/m³), Test location 18H
0.30
0.25
0.20
0.15
0.10
0.05
0.00
0.0093 µg/m³ (equivalent to acrolein odour threshold of 0.38 µg/m³)
10/06
11/06
12/06
13/06
14/06
15/06
16/06
17/06
18/06
19/06
20/06
21/06
22/06
23/06
24/06
25/06
26/06
27/06
28/06
29/06
30/06
01/07
02/07
03/07
04/07
05/07
06/07
07/07
Date

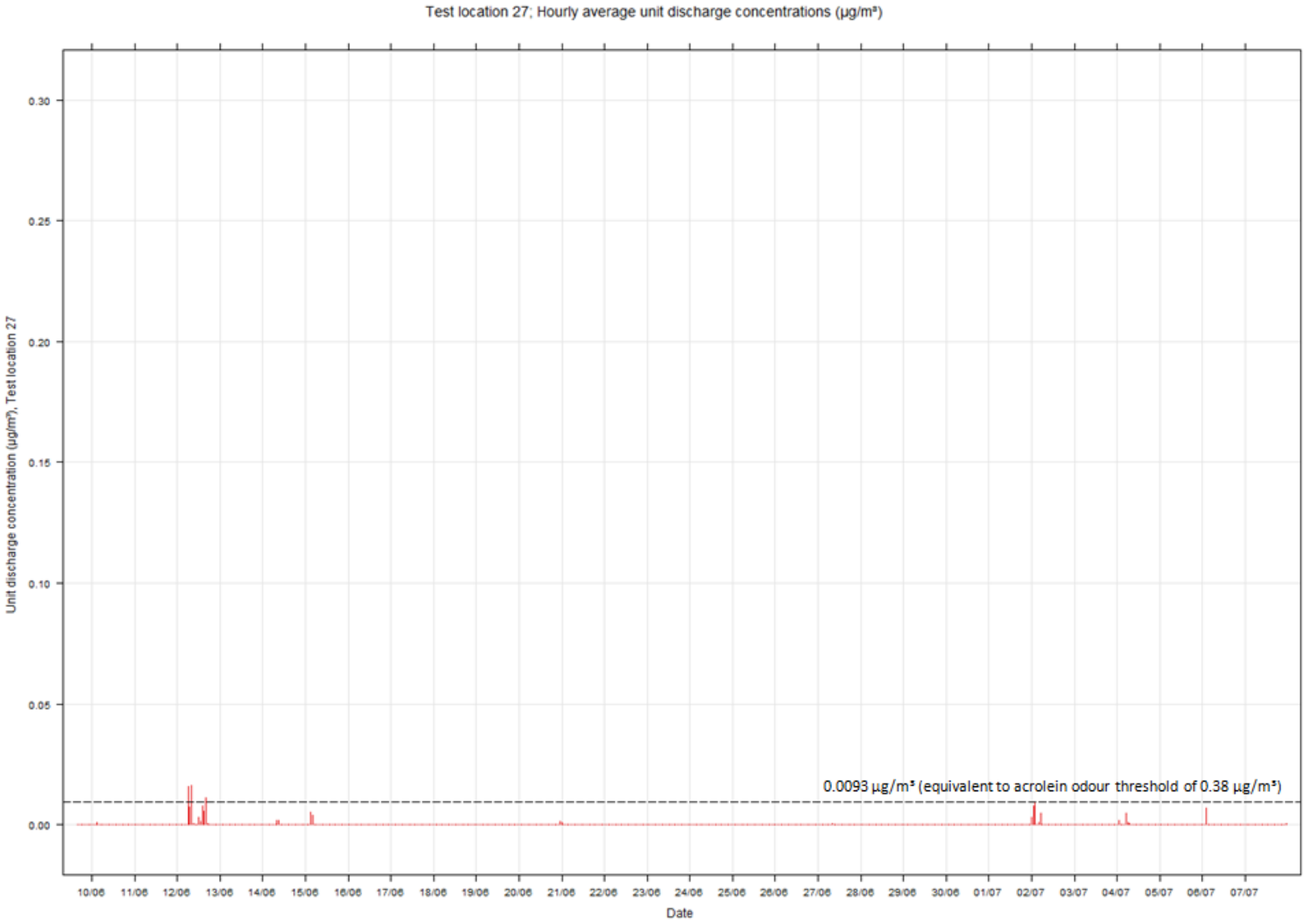

Test location 18W; Hourly average unit discharge concentrations (µg/m³)
Unit discharge concentration (µg/m³), Test location 18W
0.30
0.25
0.20
0.15
0.10
0.05
0.00
0.0093 µg/m³ (equivalent to acrolein odour threshold of 0.38 µg/m³)
10/06
11/06
12/06
13/06
14/06
15/06
16/06
17/06
18/06
19/06
20/06
21/06
22/06
23/06
24/06
25/06
26/06
27/06
28/06
29/06
30/06
01/07
02/07
03/07
04/07
05/07
06/07
07/07
Date

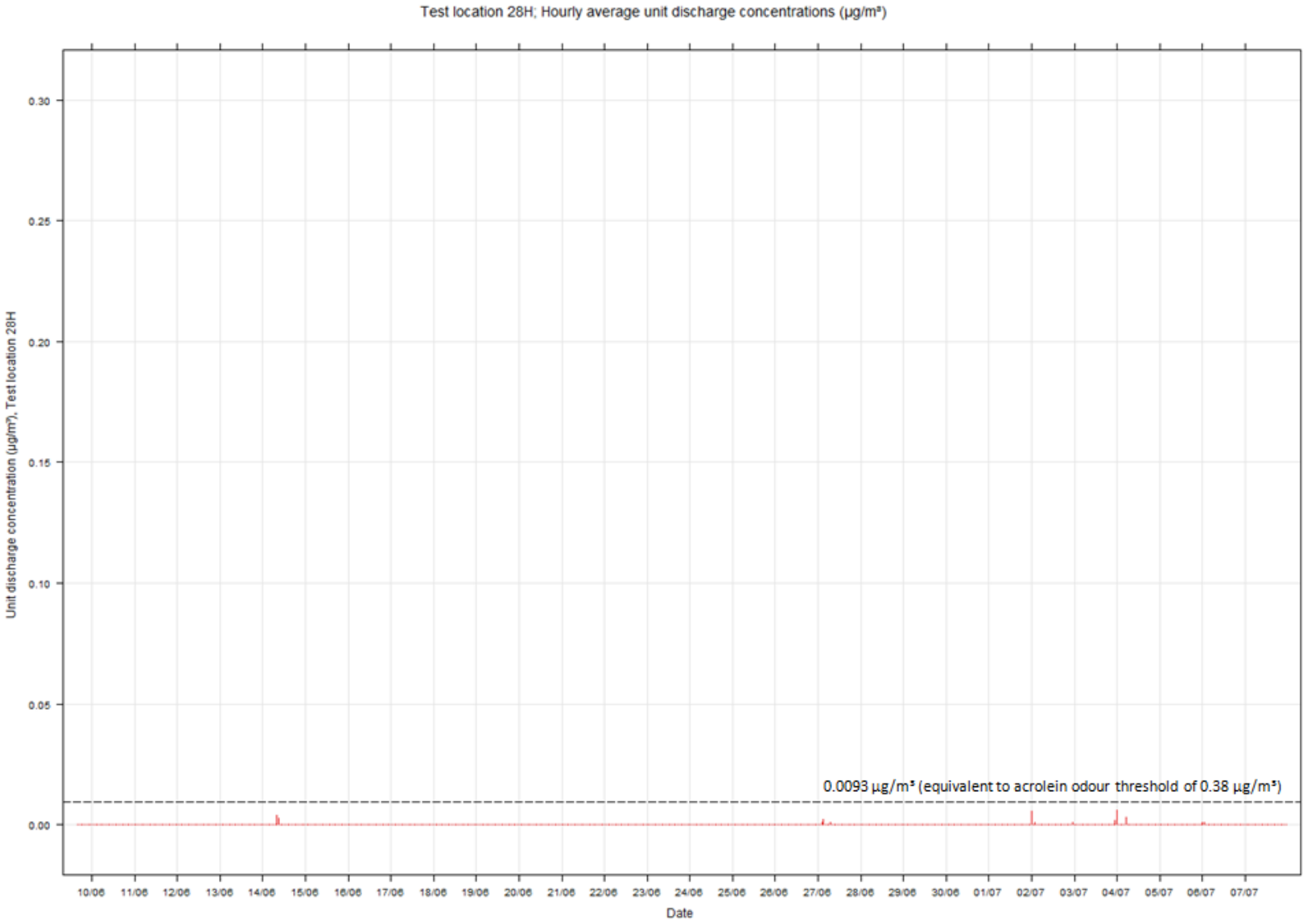

Test location 20; Hourly average unit discharge concentrations (µg/m³)
Unit discharge concentration (µg/m³), Test location 20
0.30
0.25
0.20
0.15
0.10
0.05
0.00
0.0093 µg/m³ (equivalent to acrolein odour threshold of 0.38 µg/m³)
10/06
11/06
12/06
13/06
14/06
15/06
16/06
17/06
18/06
19/06
20/06
21/06
22/06
23/06
24/06
25/06
26/06
27/06
28/06
29/06
30/06
01/07
02/07
03/07
04/07
05/07
06/07
07/07
Date

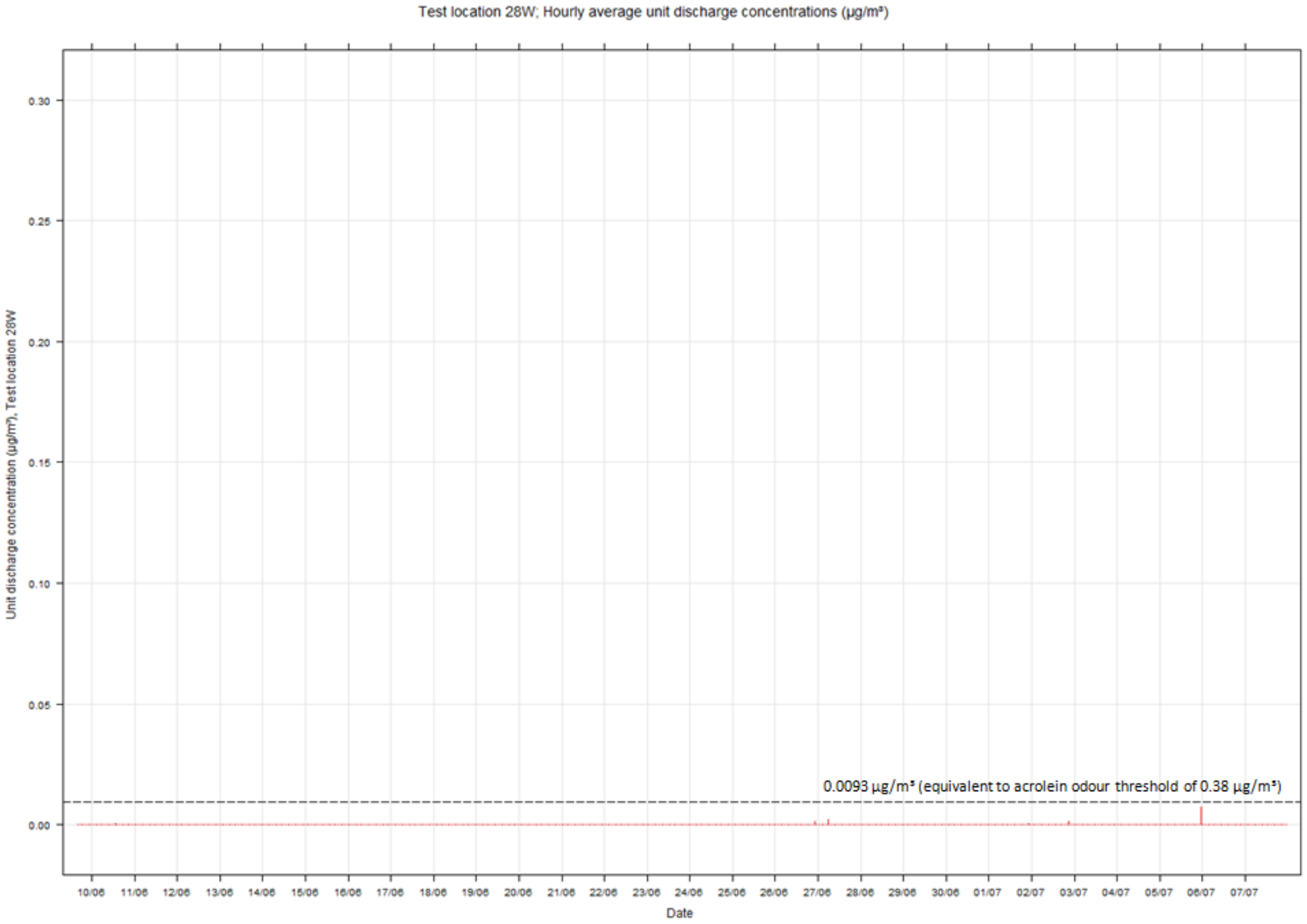

Test location 23; Hourly average unit discharge concentrations (µg/m³)
Unit discharge concentration (µg/m³), Test location 23
0.30
0.25
0.20
0.15
0.10
0.05
0.00
0.0093 µg/m³ (equivalent to acrolein odour threshold of 0.38 µg/m³)
10/06
11/06
12/06
13/06
14/06
15/06
16/06
17/06
18/06
19/06
20/06
21/06
22/06
23/06
24/06
25/06
26/06
27/06
28/06
29/06
30/06
01/07
02/07
03/07
04/07
05/07
06/07
07/07
Date

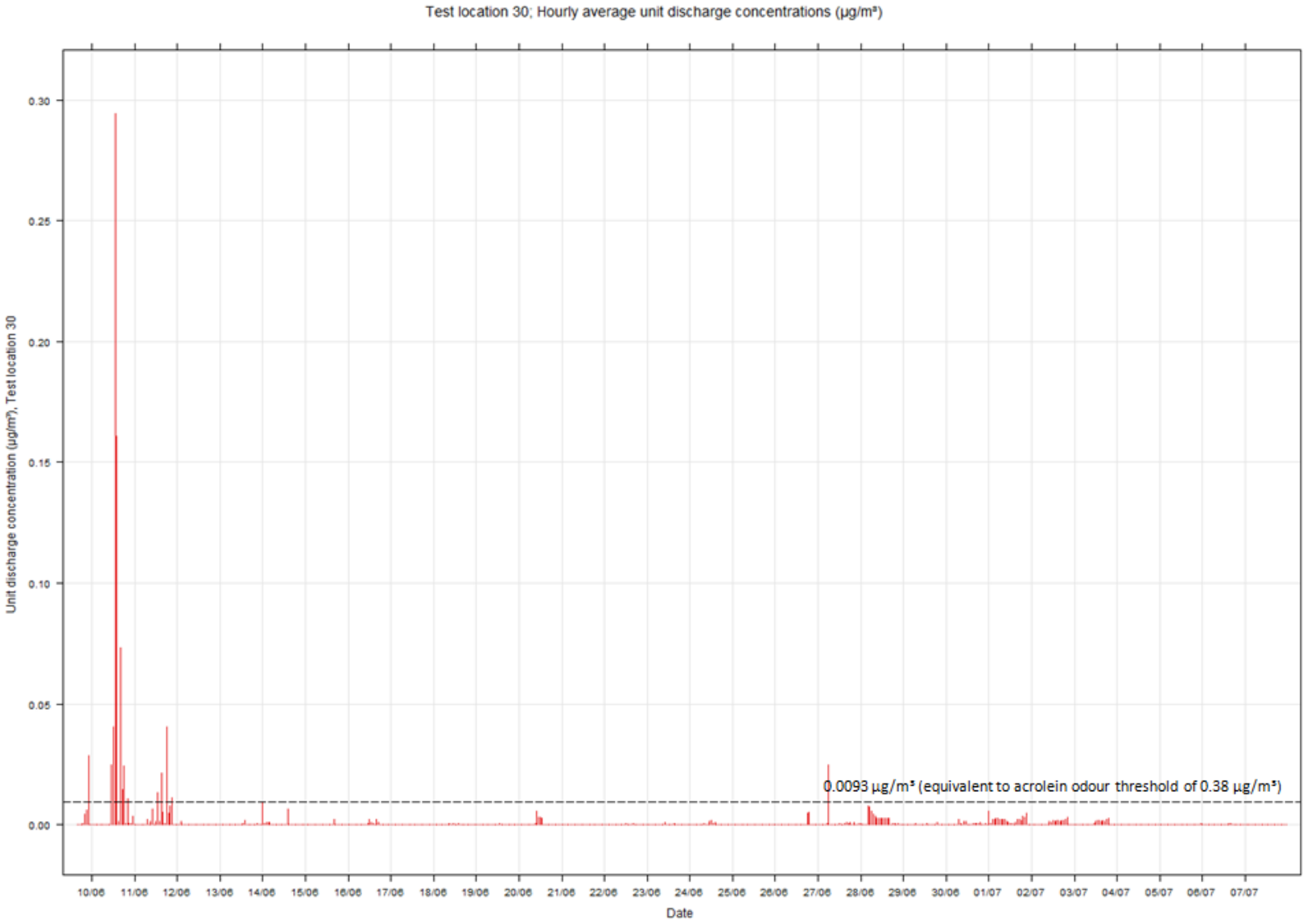

Test location 24; Hourly average unit discharge concentrations (µg/m³)
Unit discharge concentration (µg/m³), Test location 24
0.30
0.25
0.20
0.15
0.10
0.05
0.00
0.0093 µg/m³ (equivalent to acrolein odour threshold of 0.38 µg/m³)
10/06
11/06
12/06
13/06
14/06
15/06
16/06
17/06
18/06
19/06
20/06
21/06
22/06
23/06
24/06
25/06
26/06
27/06
28/06
29/06
30/06
01/07
02/07
03/07
04/07
05/07
06/07
07/07
Date

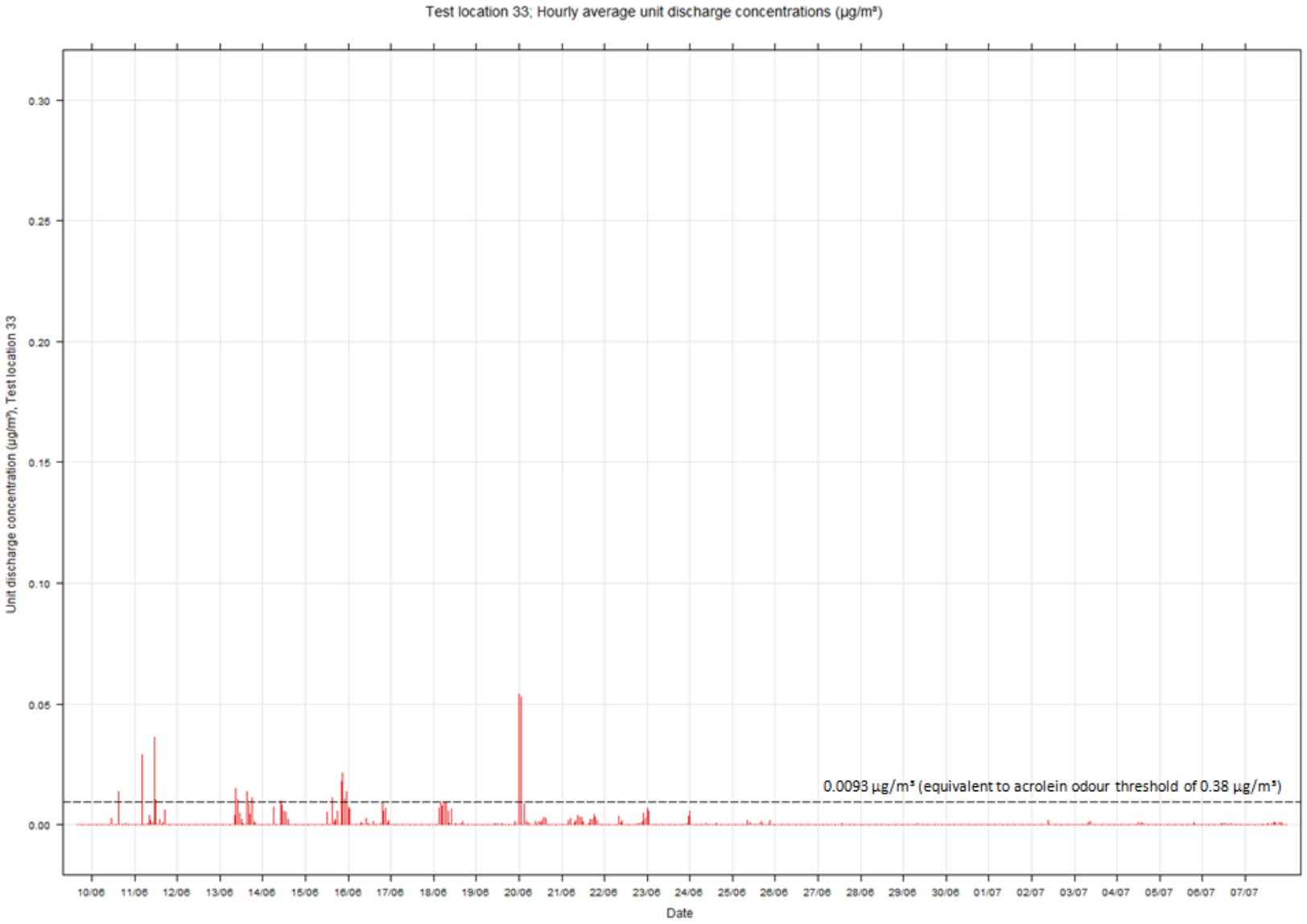

Test location 25; Hourly average unit discharge concentrations (µg/m³)
Unit discharge concentration (µg/m³), Test location 25
0.30
0.25
0.20
0.15
0.10
0.05
0.00
0.0093 µg/m³ (equivalent to acrolein odour threshold of 0.38 µg/m³)
10/06
11/06
12/06
13/06
14/06
15/06
16/06
17/06
18/06
19/06
20/06
21/06
22/06
23/06
24/06
25/06
26/06
27/06
28/06
29/06
30/06
01/07
02/07
03/07
04/07
05/07
06/07
07/07
Date

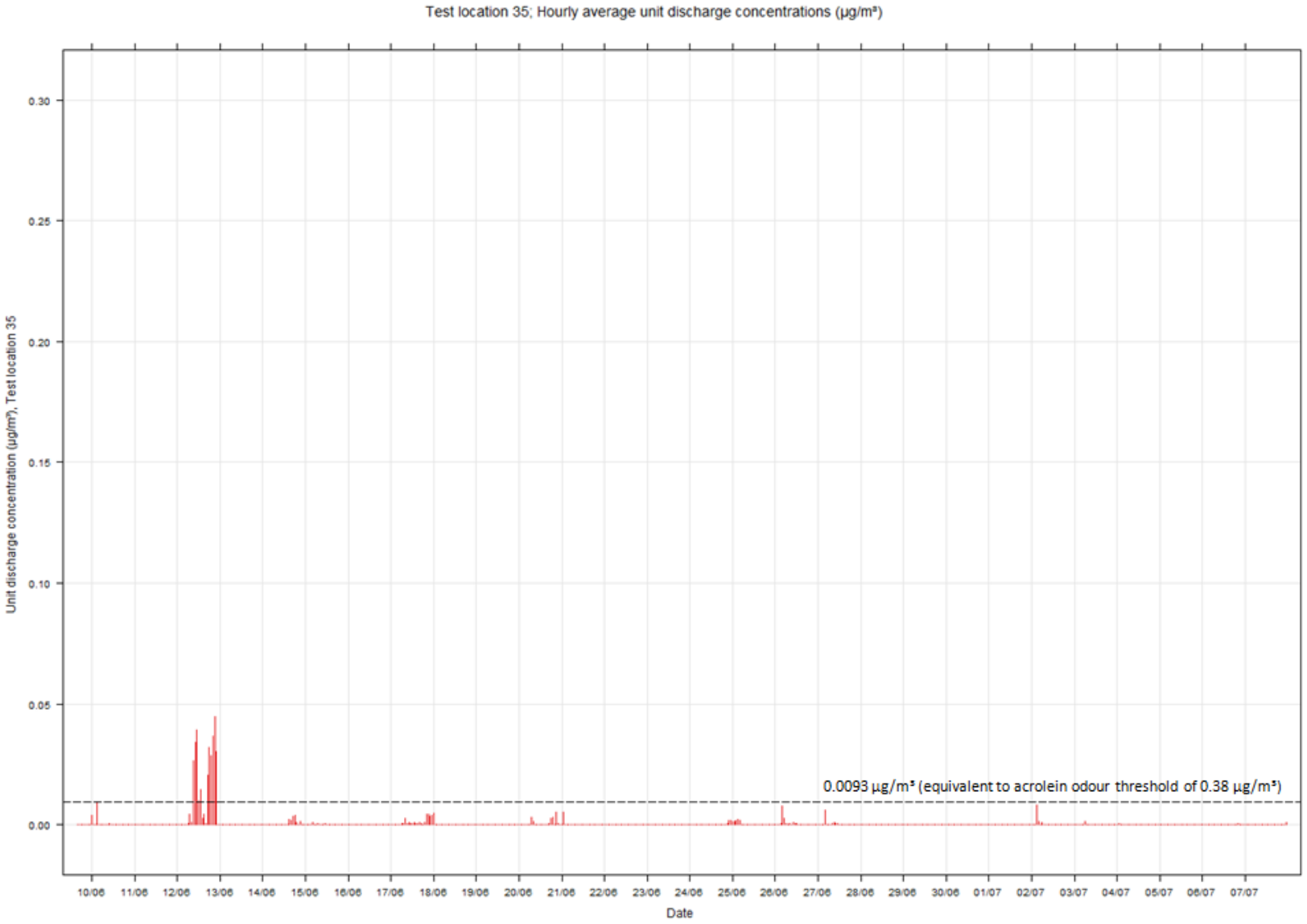

Test location 27; Hourly average unit discharge concentrations (µg/m³)
Unit discharge concentration (µg/m³), Test location 27
0.30
0.25
0.20
0.15
0.10
0.05
0.00
0.0093 µg/m³ (equivalent to acrolein odour threshold of 0.38 µg/m³)
10/06
11/06
12/06
13/06
14/06
15/06
16/06
17/06
18/06
19/06
20/06
21/06
22/06
23/06
24/06
25/06
26/06
27/06
28/06
29/06
30/06
01/07
02/07
03/07
04/07
05/07
06/07
07/07
Date

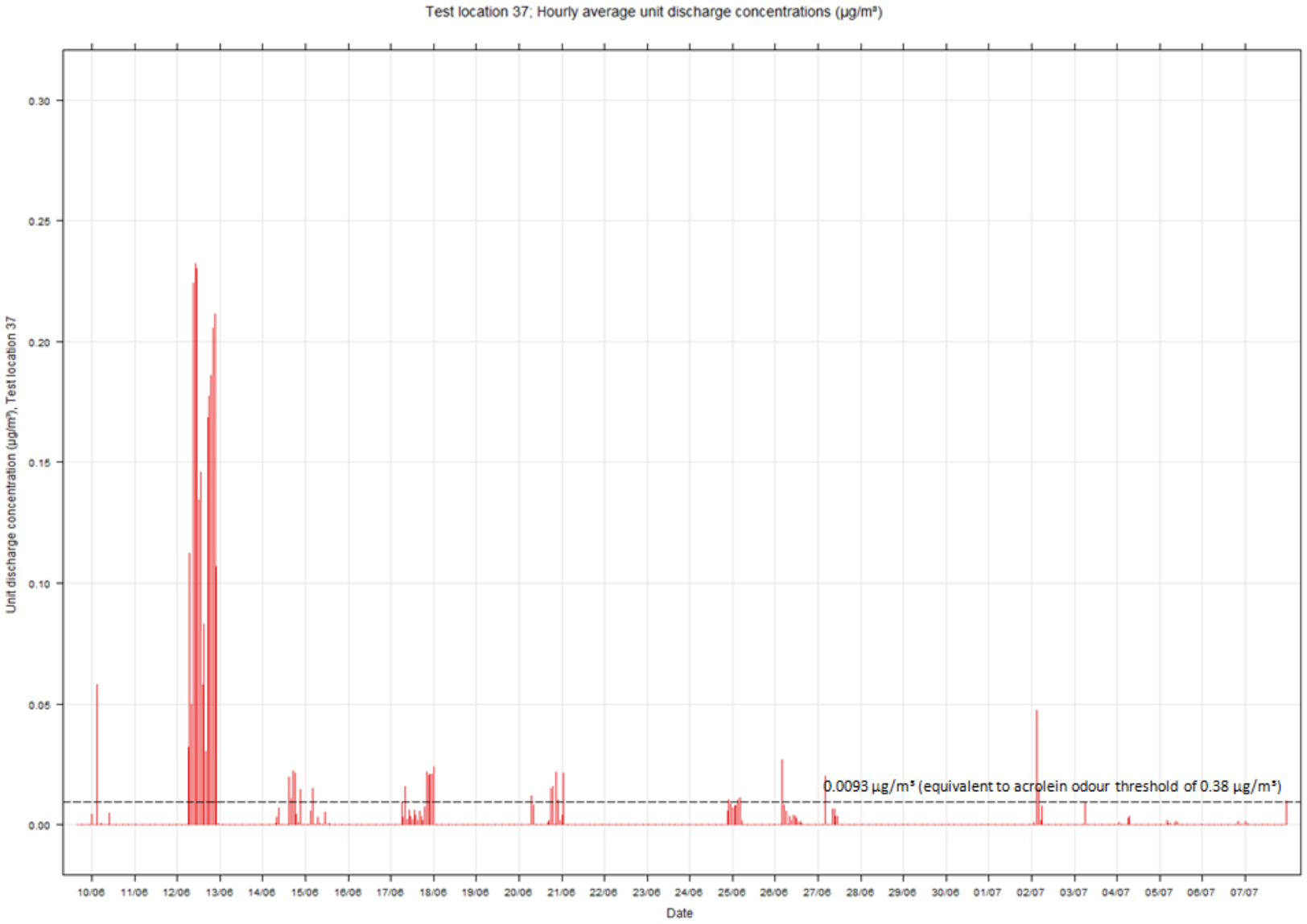

Test location 28H; Hourly average unit discharge concentrations (µg/m³)
Unit discharge concentration (µg/m³), Test location 28H
0.30
0.25
0.20
0.15
0.10
0.05
0.00
0.0093 µg/m³ (equivalent to acrolein odour threshold of 0.38 µg/m³)
10/06
11/06
12/06
13/06
14/06
15/06
16/06
17/06
18/06
19/06
20/06
21/06
22/06
23/06
24/06
25/06
26/06
27/06
28/06
29/06
30/06
01/07
02/07
03/07
04/07
05/07
06/07
07/07
Date

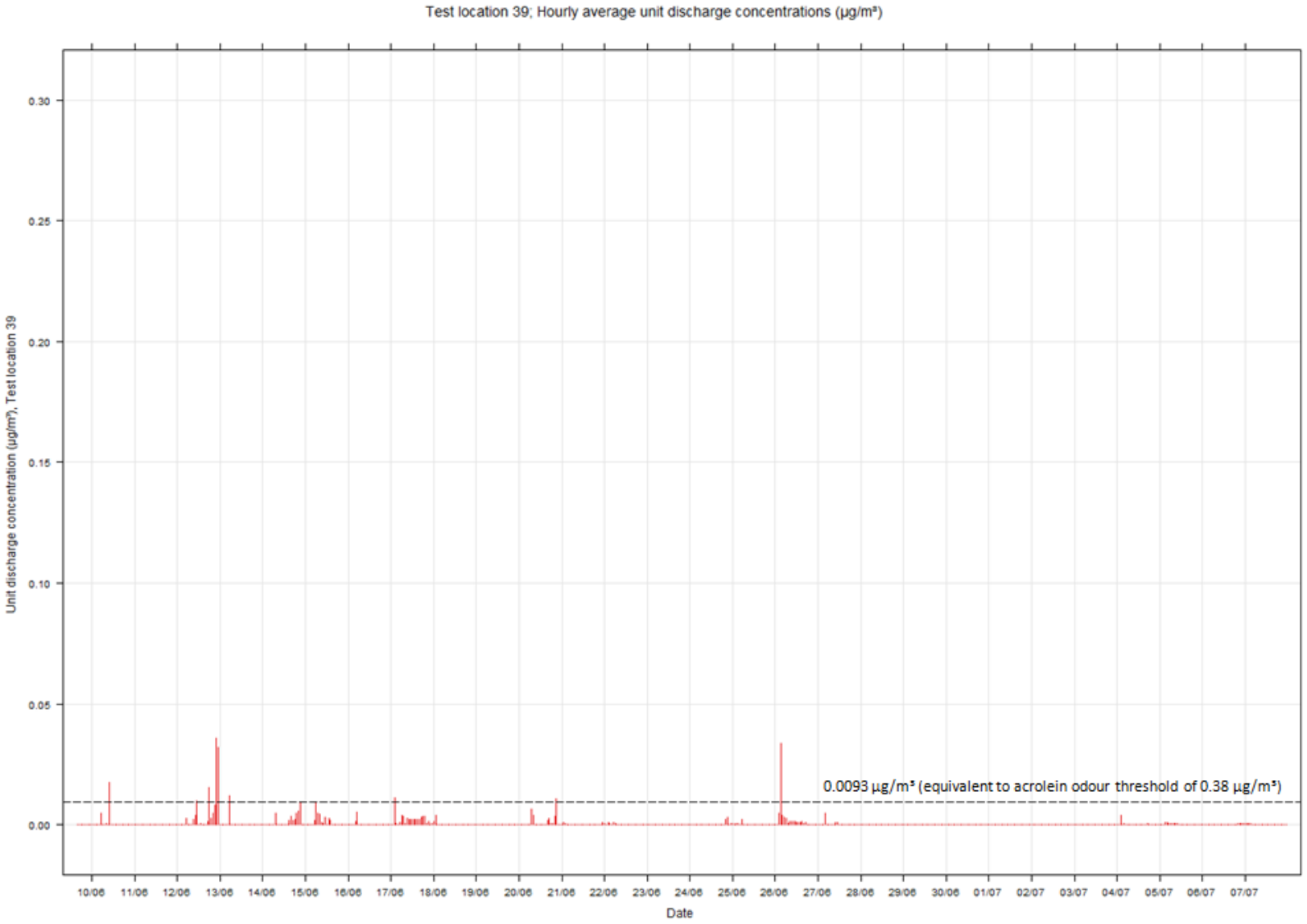

Test location 28W; Hourly average unit discharge concentrations (µg/m³)
Unit discharge concentration (µg/m³), Test location 28W
0.30
0.25
0.20
0.15
0.10
0.05
0.00
0.0093 µg/m³ (equivalent to acrolein odour threshold of 0.38 µg/m³)
10/06 11/06 12/06 13/06 14/06 15/06 16/06 17/06 18/06 19/06 20/06 21/06 22/06 23/06 24/06 25/06 26/06 27/06 28/06 29/06 30/06 01/07 02/07 03/07 04/07 05/07 06/07 07/07
Date

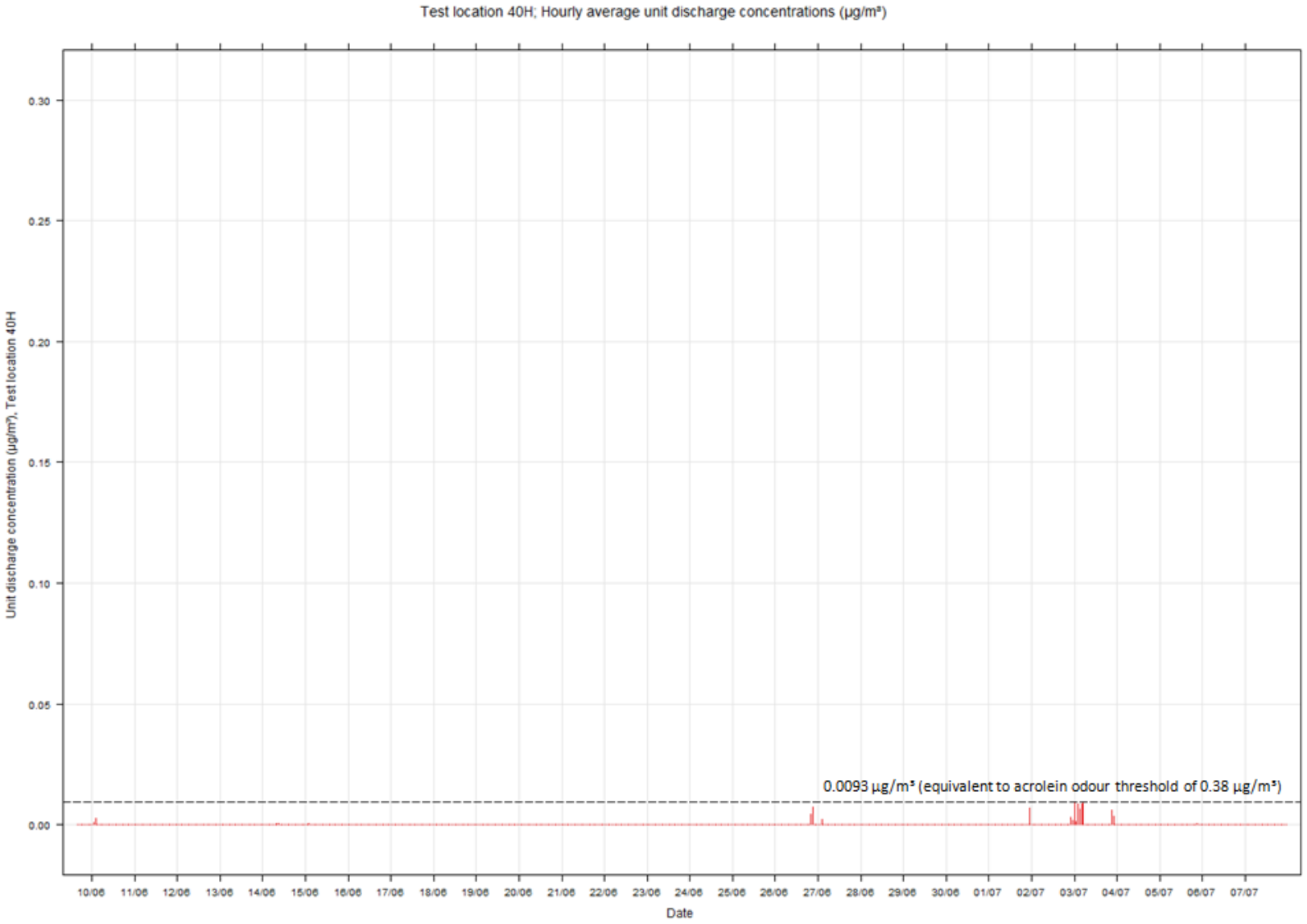

Test location 30; Hourly average unit discharge concentrations (µg/m³)
Unit discharge concentration (µg/m³), Test location 30
0.30
0.25
0.20
0.15
0.10
0.05
0.00
0.0093 µg/m³ (equivalent to acrolein odour threshold of 0.38 µg/m³)
10/06
11/06
12/06
13/06
14/06
15/06
16/06
17/06
18/06
19/06
20/06
21/06
22/06
23/06
24/06
25/06
26/06
27/06
28/06
29/06
30/06
01/07
02/07
03/07
04/07
05/07
06/07
07/07
Date

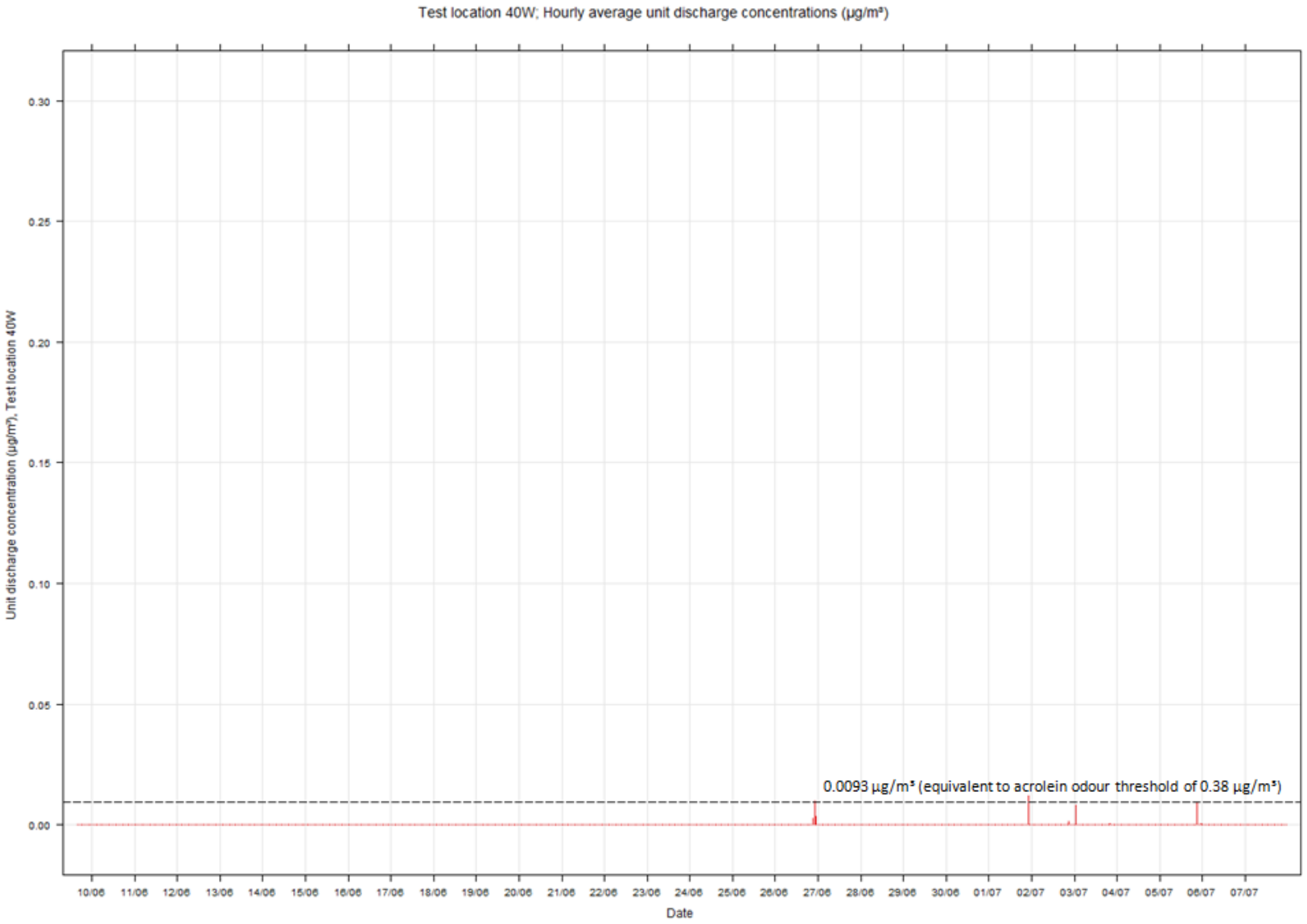

Test location 33; Hourly average unit discharge concentrations (µg/m³)
Unit discharge concentration (µg/m³), Test location 33
0.30
0.25
0.20
0.15
0.10
0.05
0.00
0.0093 µg/m³ (equivalent to acrolein odour threshold of 0.38 µg/m³)
10/06
11/06
12/06
13/06
14/06
15/06
16/06
17/06
18/06
19/06
20/06
21/06
22/06
23/06
24/06
25/06
26/06
27/06
28/06
29/06
30/06
01/07
02/07
03/07
04/07
05/07
06/07
07/07
Date


Test location 35; Hourly average unit discharge concentrations (µg/m³)
Unit discharge concentration (µg/m³), Test location 35
0.30
0.25
0.20
0.15
0.10
0.05
0.00
0.0093 µg/m³ (equivalent to acrolein odour threshold of 0.38 µg/m³)
10/06
11/06
12/06
13/06
14/06
15/06
16/06
17/06
18/06
19/06
20/06
21/06
22/06
23/06
24/06
25/06
26/06
27/06
28/06
29/06
30/06
01/07
02/07
03/07
04/07
05/07
06/07
07/07
Date


Test location 37; Hourly average unit discharge concentrations (µg/m³)
Unit discharge concentration (µg/m³), Test location 37
0.30
0.25
0.20
0.15
0.10
0.05
0.00
0.0093 µg/m³ (equivalent to acrolein odour threshold of 0.38 µg/m³)
10/06
11/06
12/06
13/06
14/06
15/06
16/06
17/06
18/06
19/06
20/06
21/06
22/06
23/06
24/06
25/06
26/06
27/06
28/06
29/06
30/06
01/07
02/07
03/07
04/07
05/07
06/07
07/07
Date


Test location 39; Hourly average unit discharge concentrations (µg/m³)
Unit discharge concentration (µg/m³), Test location 39
0.30
0.25
0.20
0.15
0.10
0.05
0.00
0.0093 µg/m³ (equivalent to acrolein odour threshold of 0.38 µg/m³)
10/06
11/06
12/06
13/06
14/06
15/06
16/06
17/06
18/06
19/06
20/06
21/06
22/06
23/06
24/06
25/06
26/06
27/06
28/06
29/06
30/06
01/07
02/07
03/07
04/07
05/07
06/07
07/07
Date


Test location 40H; Hourly average unit discharge concentrations (µg/m³)
Unit discharge concentration (µg/m³), Test location 40H
0.30
0.25
0.20
0.15
0.10
0.05
0.00
0.0093 µg/m³ (equivalent to acrolein odour threshold of 0.38 µg/m³)
10/06
11/06
12/06
13/06
14/06
15/06
16/06
17/06
18/06
19/06
20/06
21/06
22/06
23/06
24/06
25/06
26/06
27/06
28/06
29/06
30/06
01/07
02/07
03/07
04/07
05/07
06/07
07/07
Date


Test location 40W; Hourly average unit discharge concentrations (µg/m³)
Unit discharge concentration (µg/m³), Test location 40W
0.30
0.25
0.20
0.15
0.10
0.05
0.00
0.0093 µg/m³ (equivalent to acrolein odour threshold of 0.38 µg/m³)
10/06 11/06 12/06 13/06 14/06 15/06 16/06 17/06 18/06 19/06 20/06 21/06 22/06 23/06 24/06 25/06 26/06 27/06 28/06 29/06 30/06 01/07 02/07 03/07 04/07 05/07 06/07 07/07
Date

Sonae GLO: Further Modelling

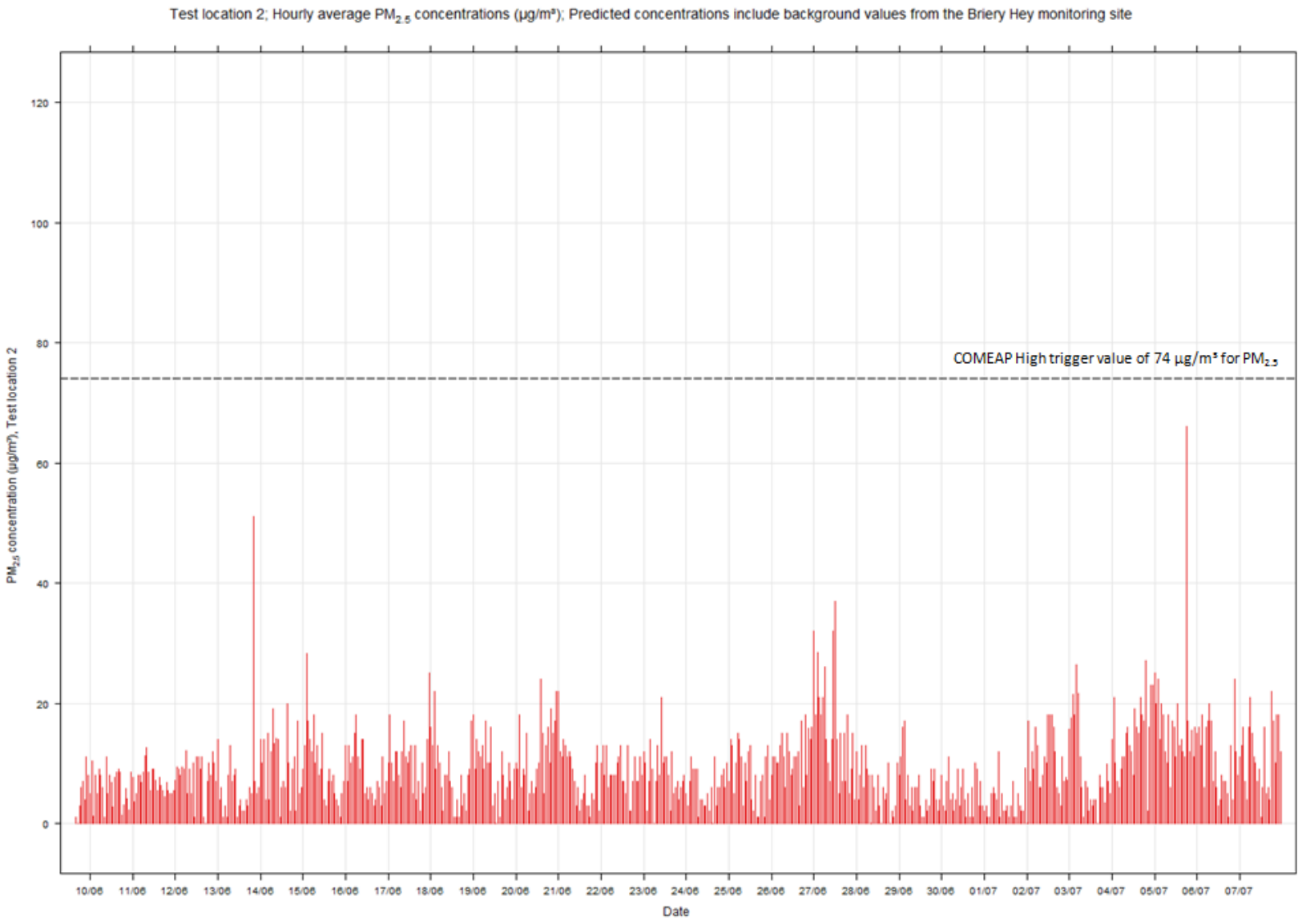
5. Histograms of modelled hourly concentrations for the 20 Test Claimants

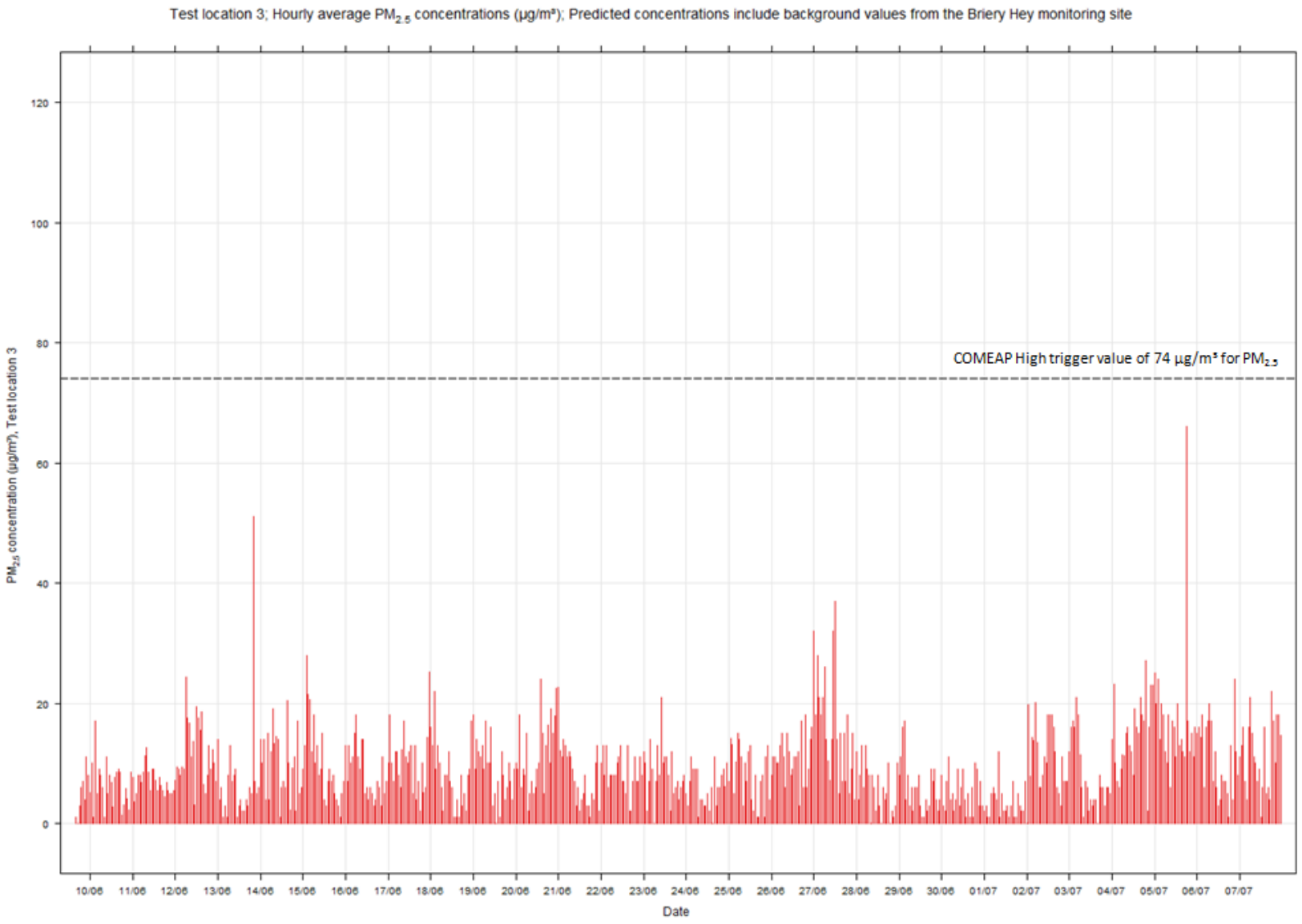
iii) *Modelled $PM_{2.5}$ concentrations compared against the COMEAP High trigger value of 74 $\mu g/m^3$*

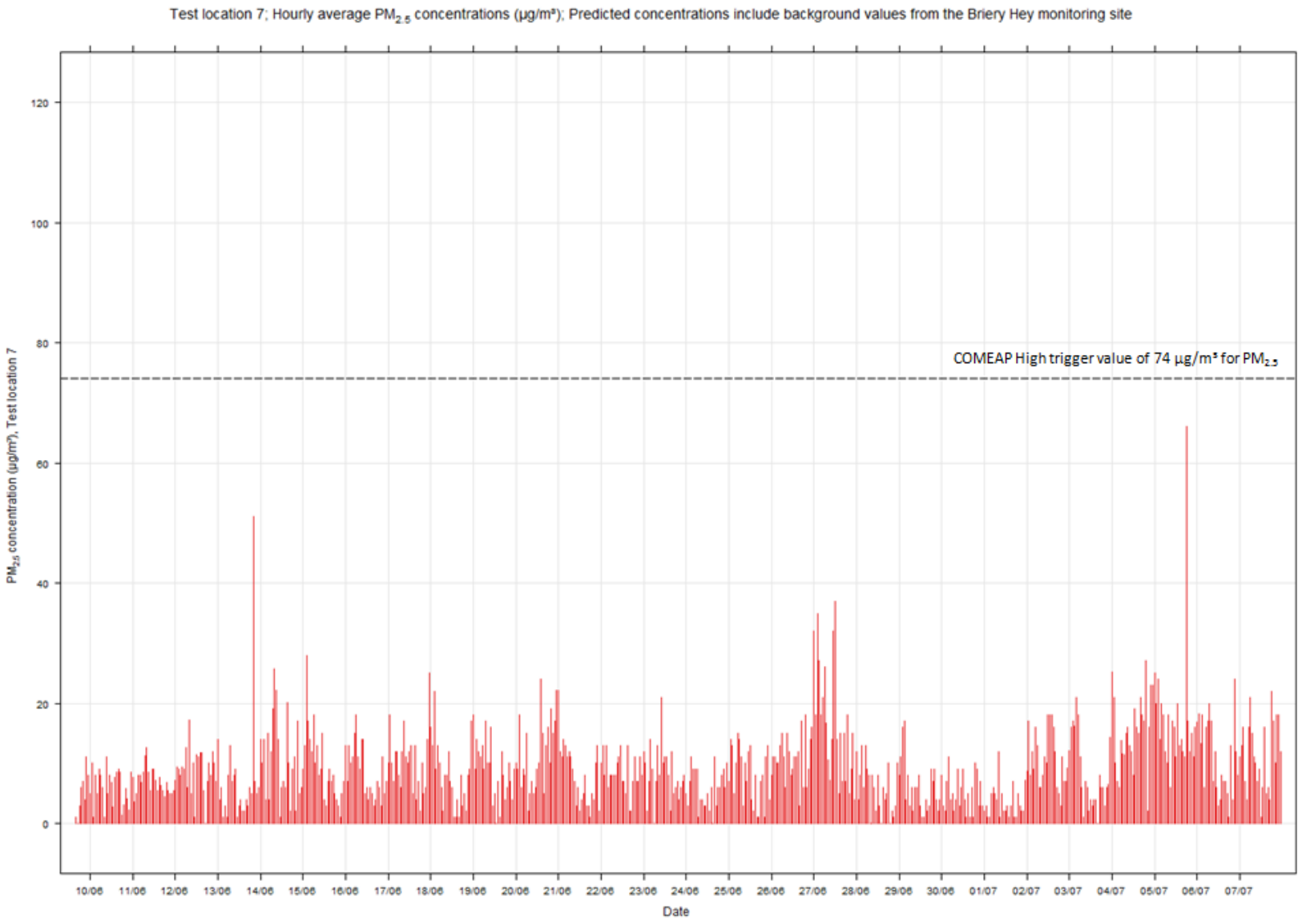
The 23 time series histograms in this document show modelled hourly average concentrations of $PM_{2.5}$ at the locations of the 20 Test Claimants. Modelled concentrations include the modelled contribution from the fire and background concentrations using measurements from the Briery Hey monitoring site.

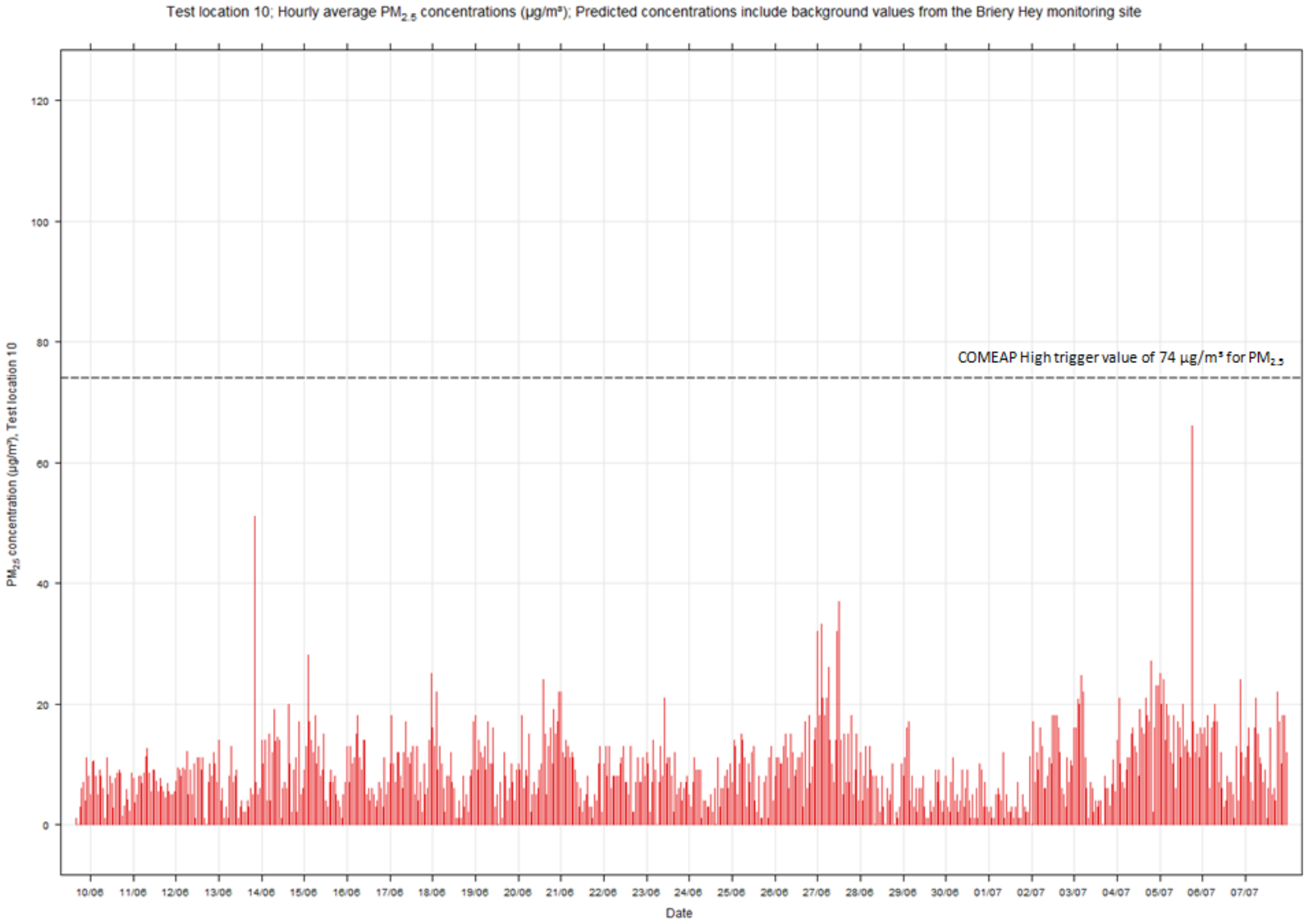
The $PM_{2.5}$ measurements from Briery Hey contain a 43-hour period between the 10th June 2011 and 12th June 2011 where no $PM_{2.5}$ data are recorded. For this period, $PM_{2.5}$ concentrations have been estimated from PM_{10} concentrations assuming a $PM_{2.5}/PM_{10}$ ratio of 0.45. This ratio is based on the ratio of mean $PM_{2.5}$ and PM_{10} concentrations over the period of the fire where both PM_{10} and $PM_{2.5}$ concentrations are recorded at Briery Hey.

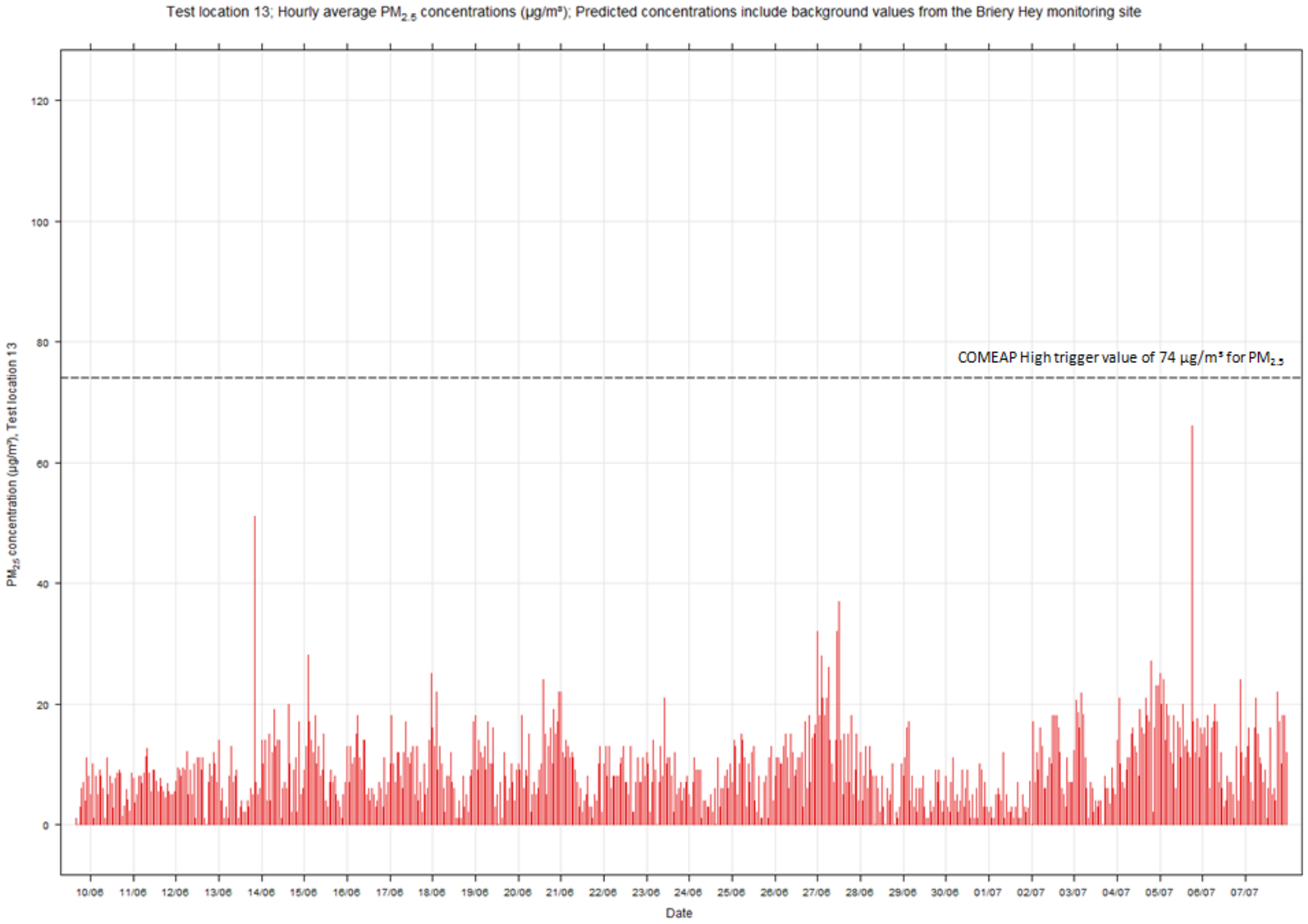
This methodology for estimating $PM_{2.5}$ concentrations for the period of missing data was agreed between CERC and Envirobods

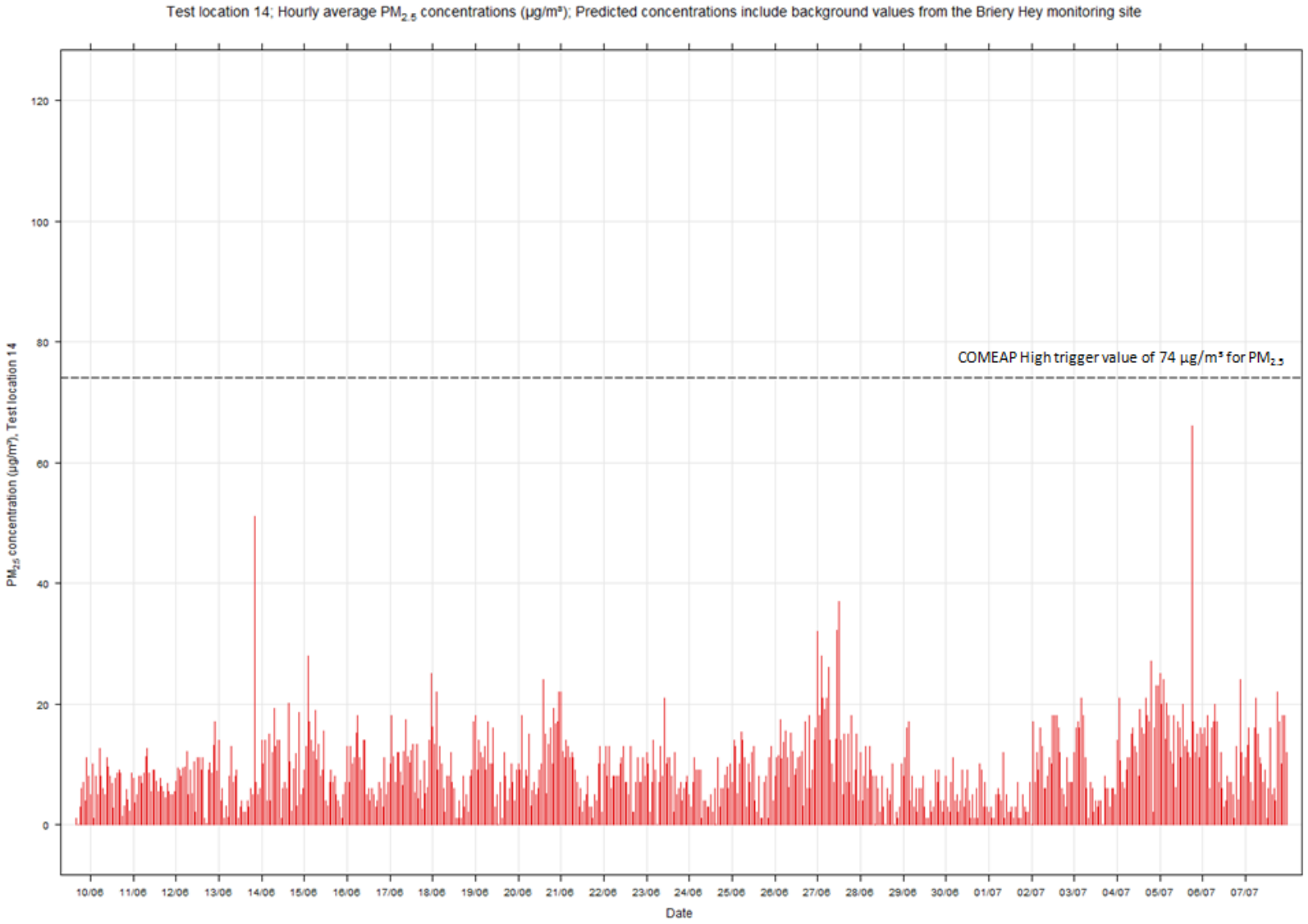

Test location 2; Hourly average PM2.5 concentrations (µg/m³); Predicted concentrations include background values from the Briery Hey monitoring site
PM2.5 concentration (µg/m³), Test location 2
120
100
80
60
40
20
0
COMEAP High trigger value of 74 µg/m³ for PM2.5
10/06
11/06
12/06
13/06
14/06
15/06
16/06
17/06
18/06
19/06
20/06
21/06
22/06
23/06
24/06
25/06
26/06
27/06
28/06
29/06
30/06
01/07
02/07
03/07
04/07
05/07
06/07
07/07
Date

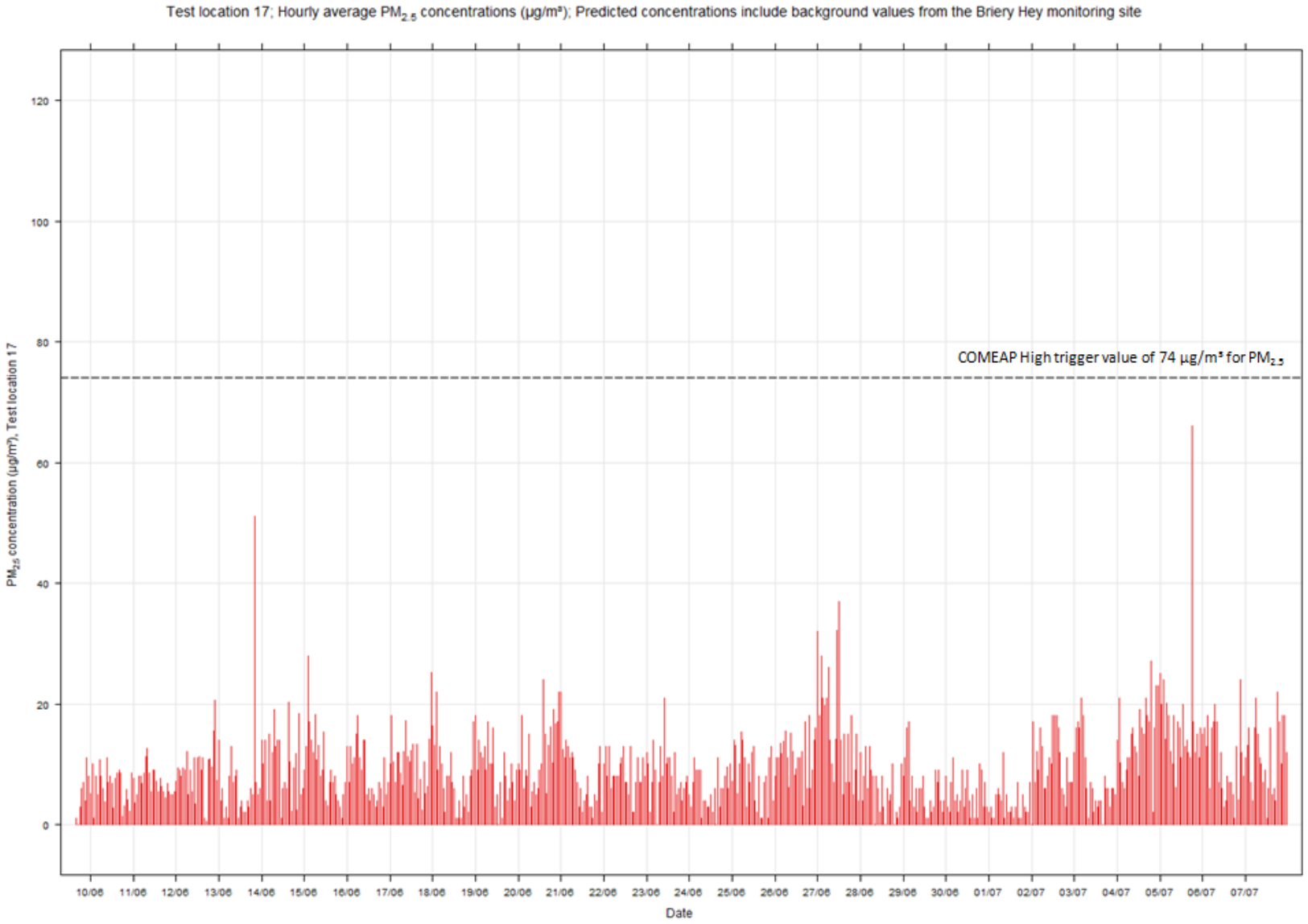

Test location 3; Hourly average PM2.5 concentrations (µg/m³); Predicted concentrations include background values from the Briery Hey monitoring site
PM2.5 concentration (µg/m³), Test location 3
120
100
80
60
40
20
0
COMEAP High trigger value of 74 µg/m³ for PM2.5
10/06
11/06
12/06
13/06
14/06
15/06
16/06
17/06
18/06
19/06
20/06
21/06
22/06
23/06
24/06
25/06
26/06
27/06
28/06
29/06
30/06
01/07
02/07
03/07
04/07
05/07
06/07
07/07
Date

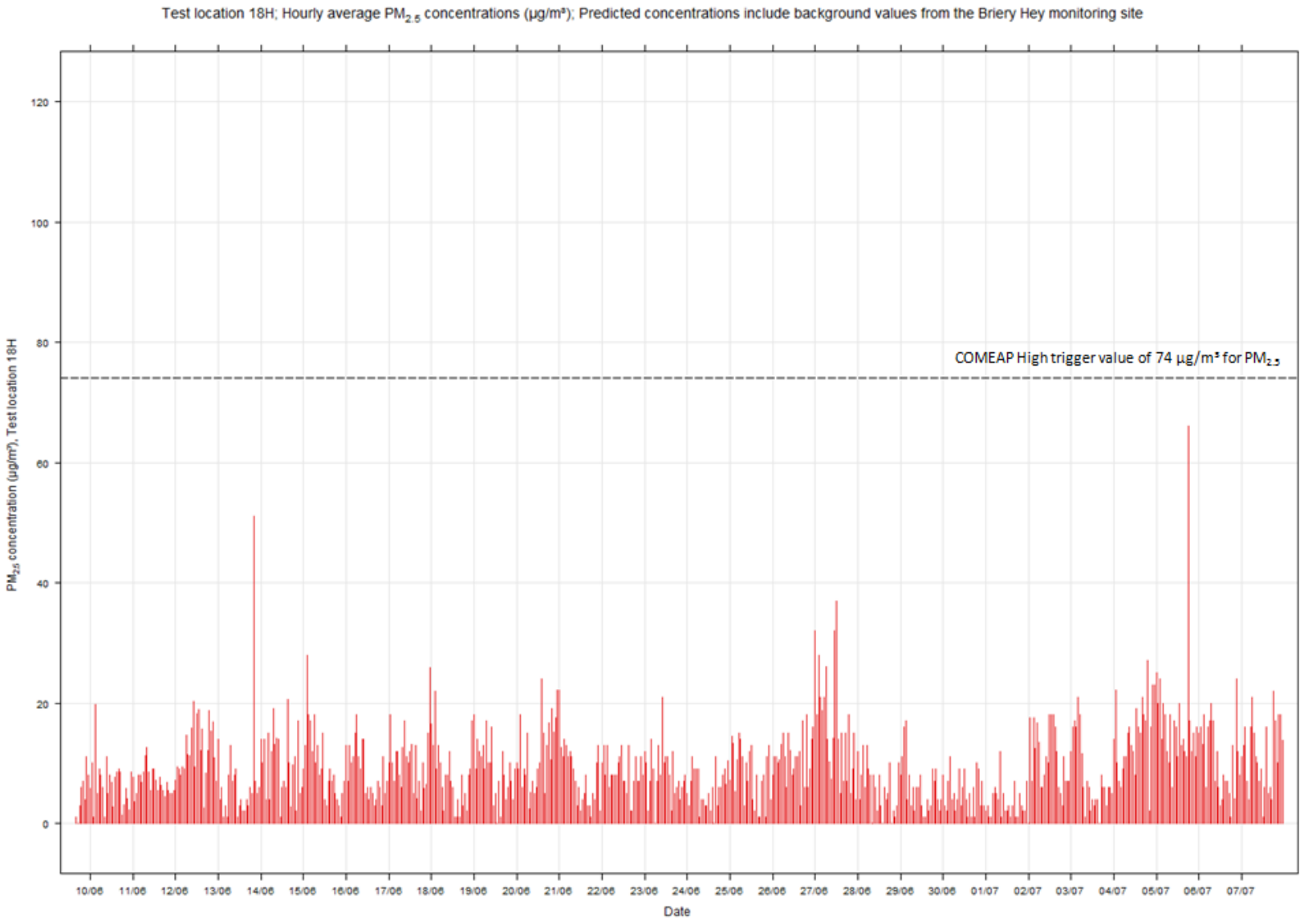

Test location 7; Hourly average PM2.5 concentrations (µg/m³); Predicted concentrations include background values from the Briery Hey monitoring site
PM2.5 concentration (µg/m³), Test location 7
120
100
80
60
40
20
0
COMEAP High trigger value of 74 µg/m³ for PM2.5
10/06
11/06
12/06
13/06
14/06
15/06
16/06
17/06
18/06
19/06
20/06
21/06
22/06
23/06
24/06
25/06
26/06
27/06
28/06
29/06
30/06
01/07
02/07
03/07
04/07
05/07
06/07
07/07
Date

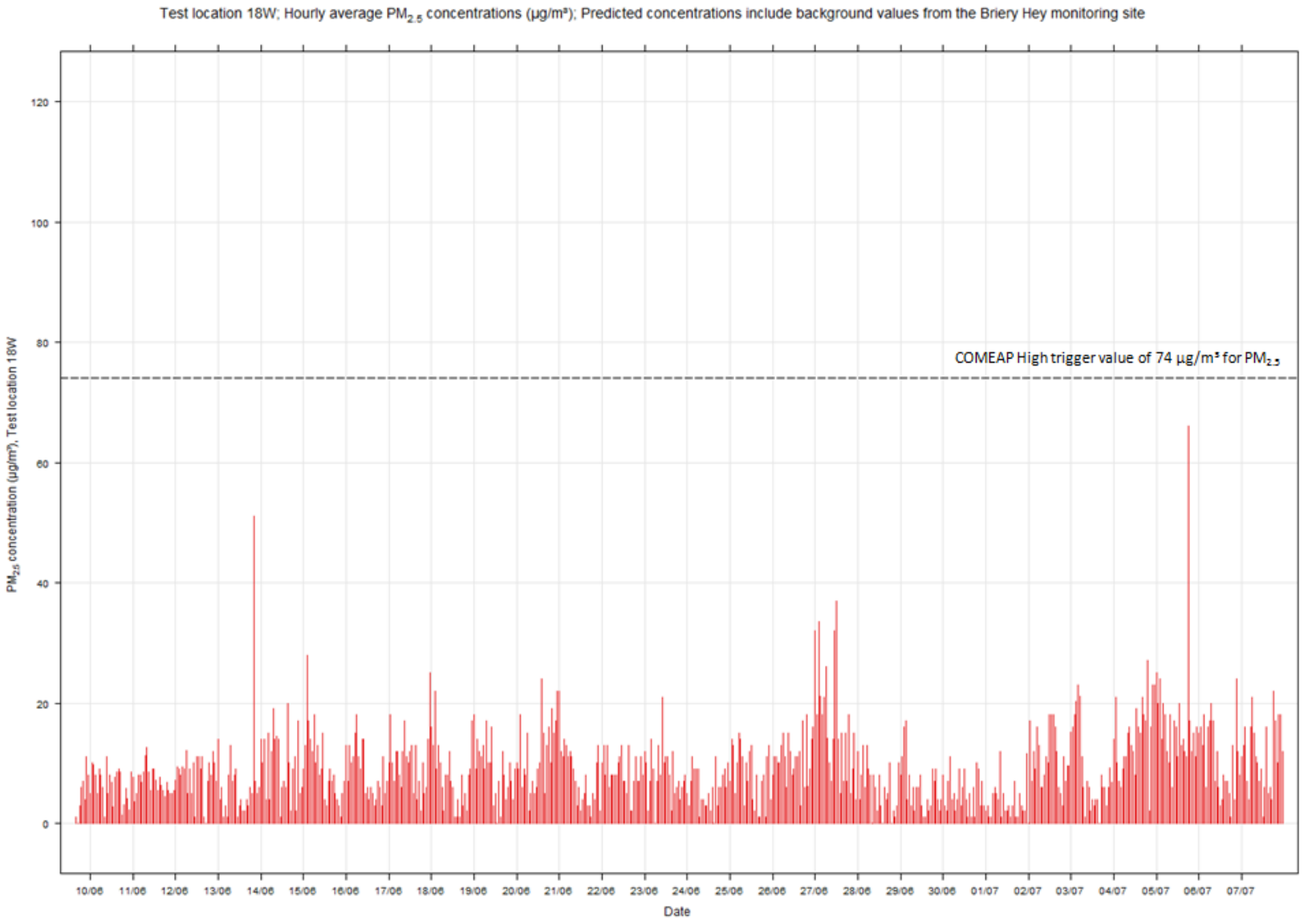

Test location 10; Hourly average PM2.5 concentrations (µg/m³); Predicted concentrations include background values from the Briery Hey monitoring site
PM2.5 concentration (µg/m³), Test location 10
120
100
80
60
40
20
0
COMEAP High trigger value of 74 µg/m³ for PM2.5
10/06
11/06
12/06
13/06
14/06
15/06
16/06
17/06
18/06
19/06
20/06
21/06
22/06
23/06
24/06
25/06
26/06
27/06
28/06
29/06
30/06
01/07
02/07
03/07
04/07
05/07
06/07
07/07
Date

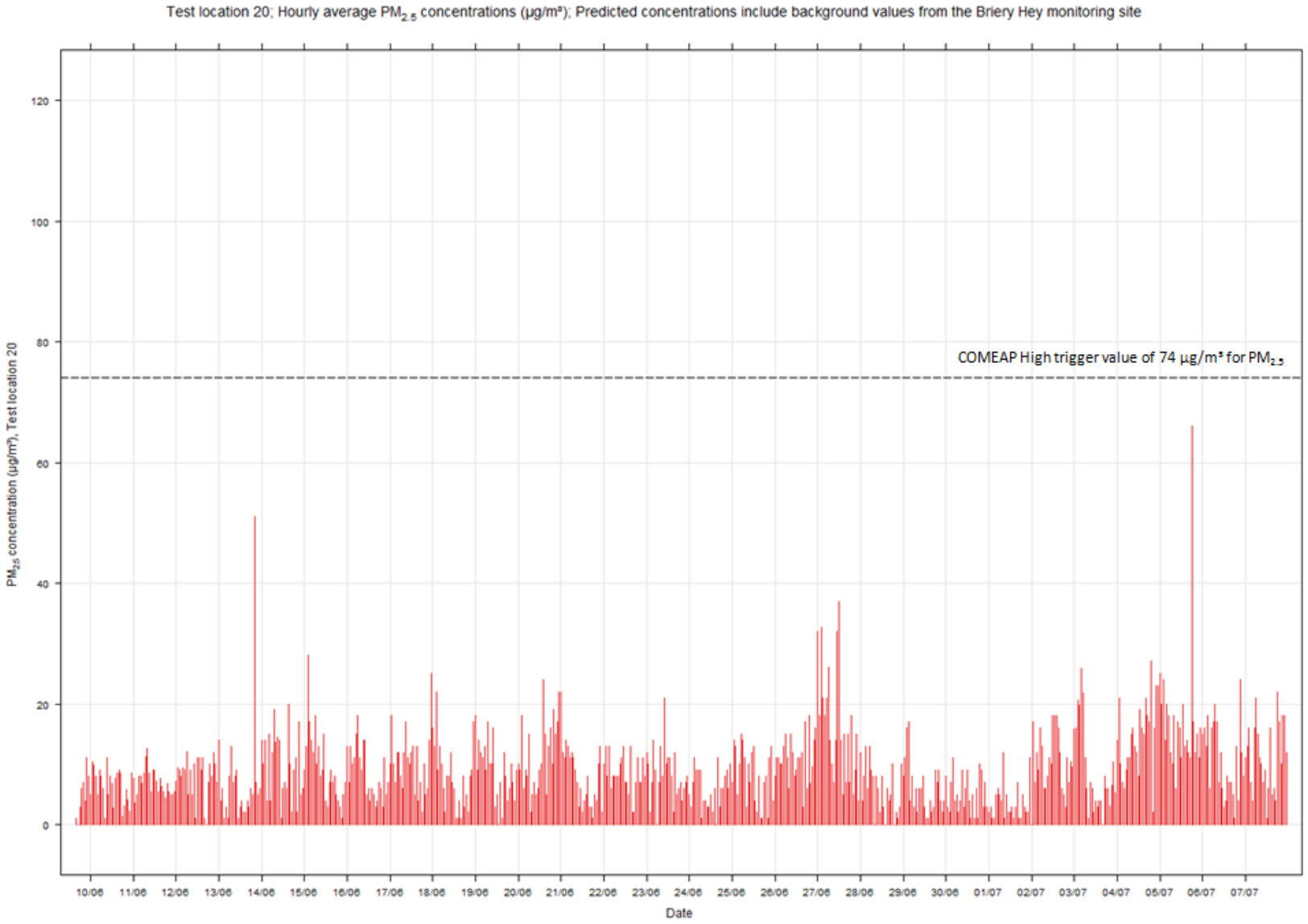

Test location 13; Hourly average PM2.5 concentrations (µg/m³); Predicted concentrations include background values from the Briery Hey monitoring site
PM2.5 concentration (µg/m³), Test location 13
120
100
80
60
40
20
0
COMEAP High trigger value of 74 µg/m³ for PM2.5
10/06
11/06
12/06
13/06
14/06
15/06
16/06
17/06
18/06
19/06
20/06
21/06
22/06
23/06
24/06
25/06
26/06
27/06
28/06
29/06
30/06
01/07
02/07
03/07
04/07
05/07
06/07
07/07
Date

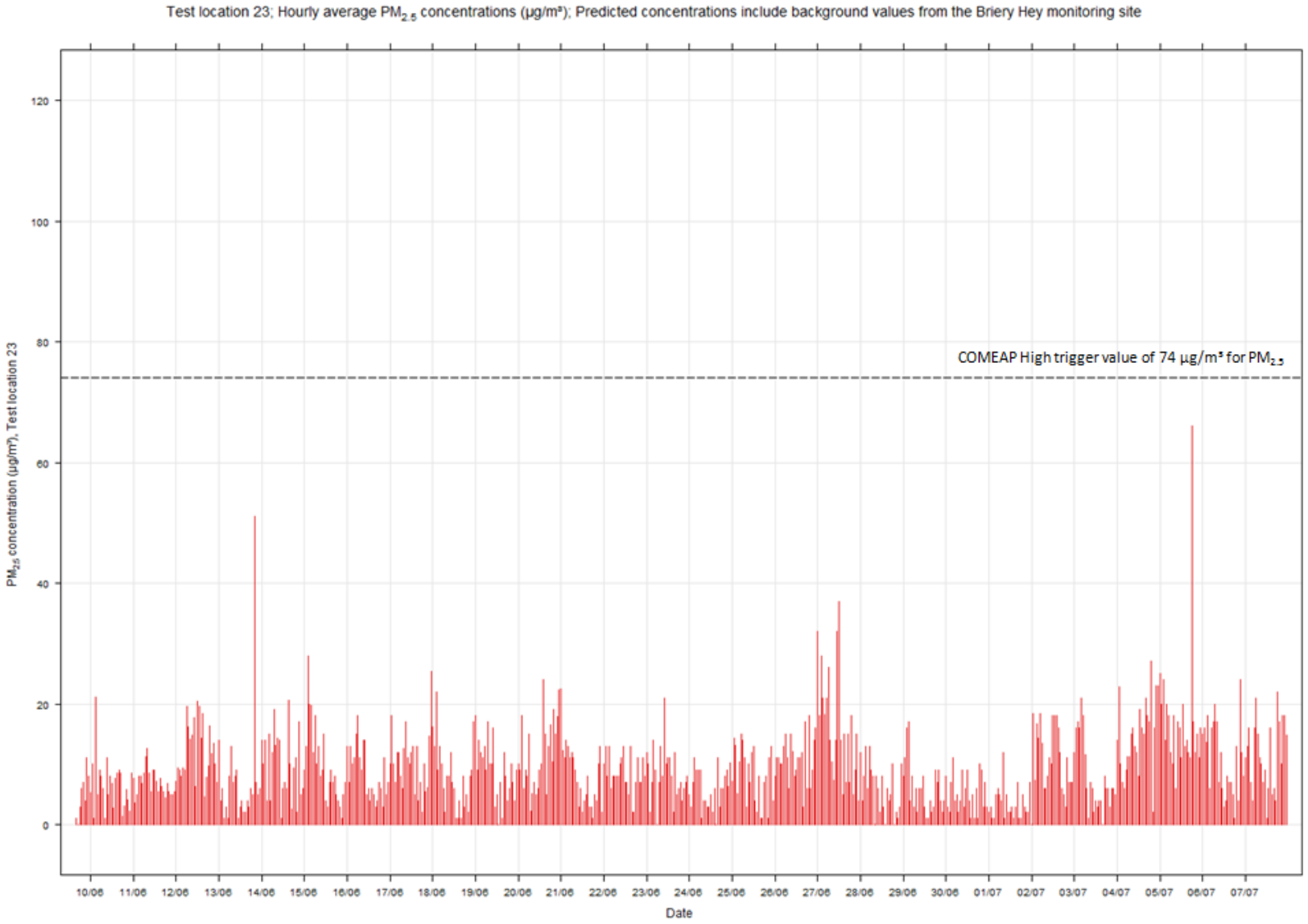

Test location 14; Hourly average PM2.5 concentrations (µg/m³); Predicted concentrations include background values from the Briery Hey monitoring site
COMEAP High trigger value of 74 µg/m³ for PM2.5
PM2.5 concentration (µg/m³), Test location 14
0
20
40
60
80
100
120
10/06
11/06
12/06
13/06
14/06
15/06
16/06
17/06
18/06
19/06
20/06
21/06
22/06
23/06
24/06
25/06
26/06
27/06
28/06
29/06
30/06
01/07
02/07
03/07
04/07
05/07
06/07
07/07
Date

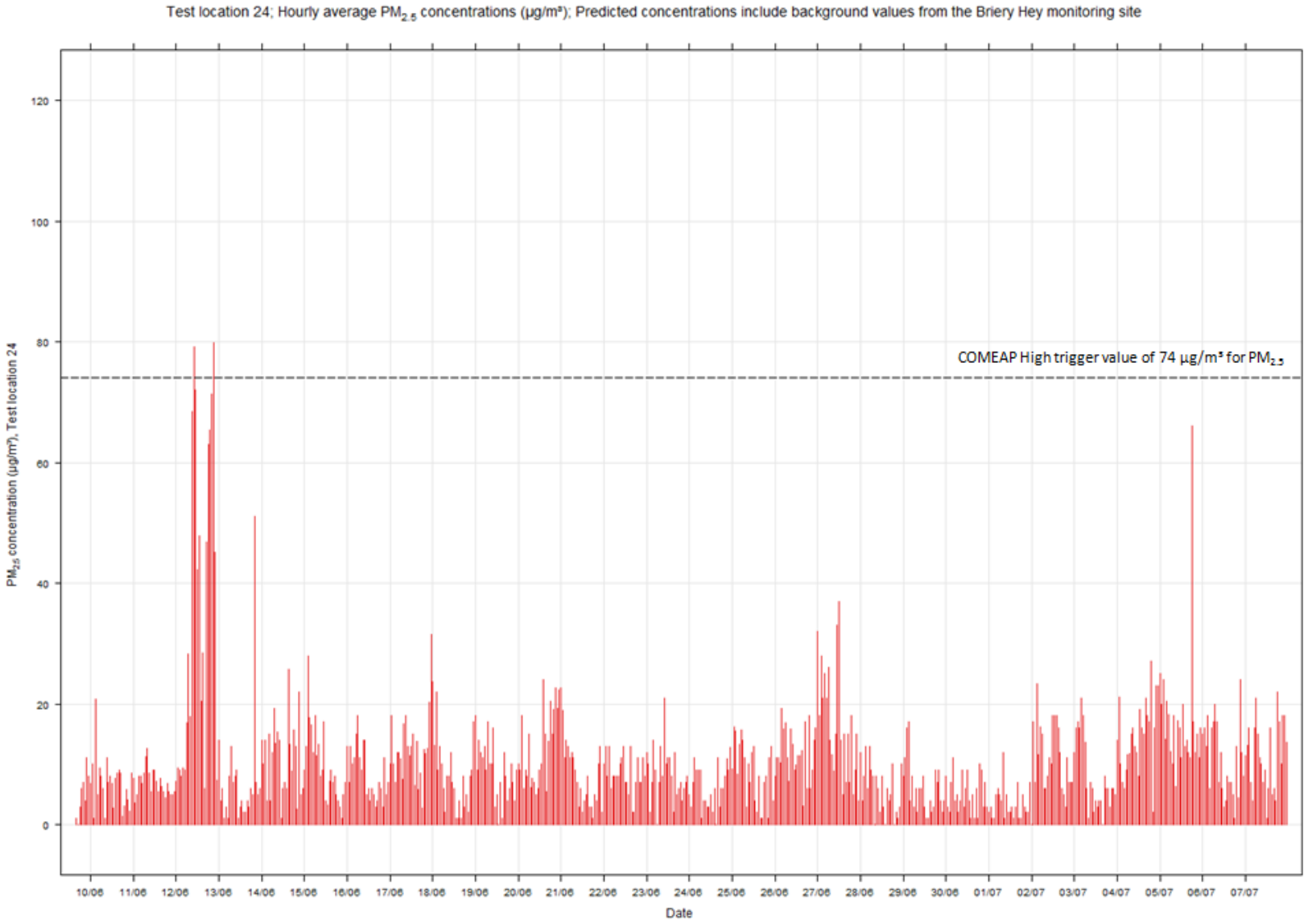

Test location 17; Hourly average PM2.5 concentrations (µg/m³); Predicted concentrations include background values from the Briery Hey monitoring site
COMEAP High trigger value of 74 µg/m³ for PM2.5
PM2.5 concentration (µg/m³), Test location 17
0
20
40
60
80
100
120
10/06
11/06
12/06
13/06
14/06
15/06
16/06
17/06
18/06
19/06
20/06
21/06
22/06
23/06
24/06
25/06
26/06
27/06
28/06
29/06
30/06
01/07
02/07
03/07
04/07
05/07
06/07
07/07
Date

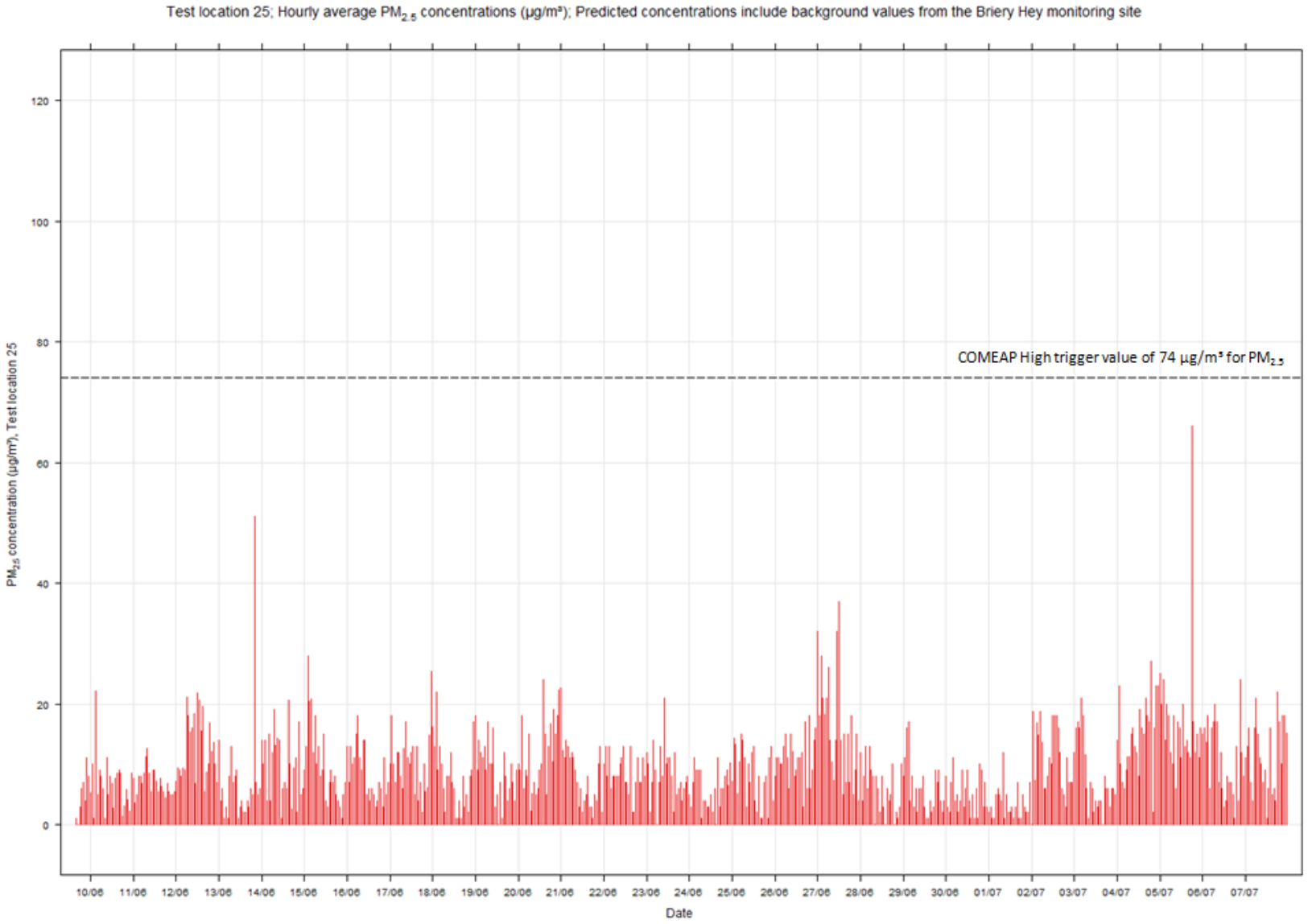

Test location 18H; Hourly average PM2.5 concentrations (µg/m³); Predicted concentrations include background values from the Briery Hey monitoring site
PM2.5 concentration (µg/m³), Test location 18H
120
100
80
60
40
20
0
COMEAP High trigger value of 74 µg/m³ for PM2.5
10/06
11/06
12/06
13/06
14/06
15/06
16/06
17/06
18/06
19/06
20/06
21/06
22/06
23/06
24/06
25/06
26/06
27/06
28/06
29/06
30/06
01/07
02/07
03/07
04/07
05/07
06/07
07/07
Date

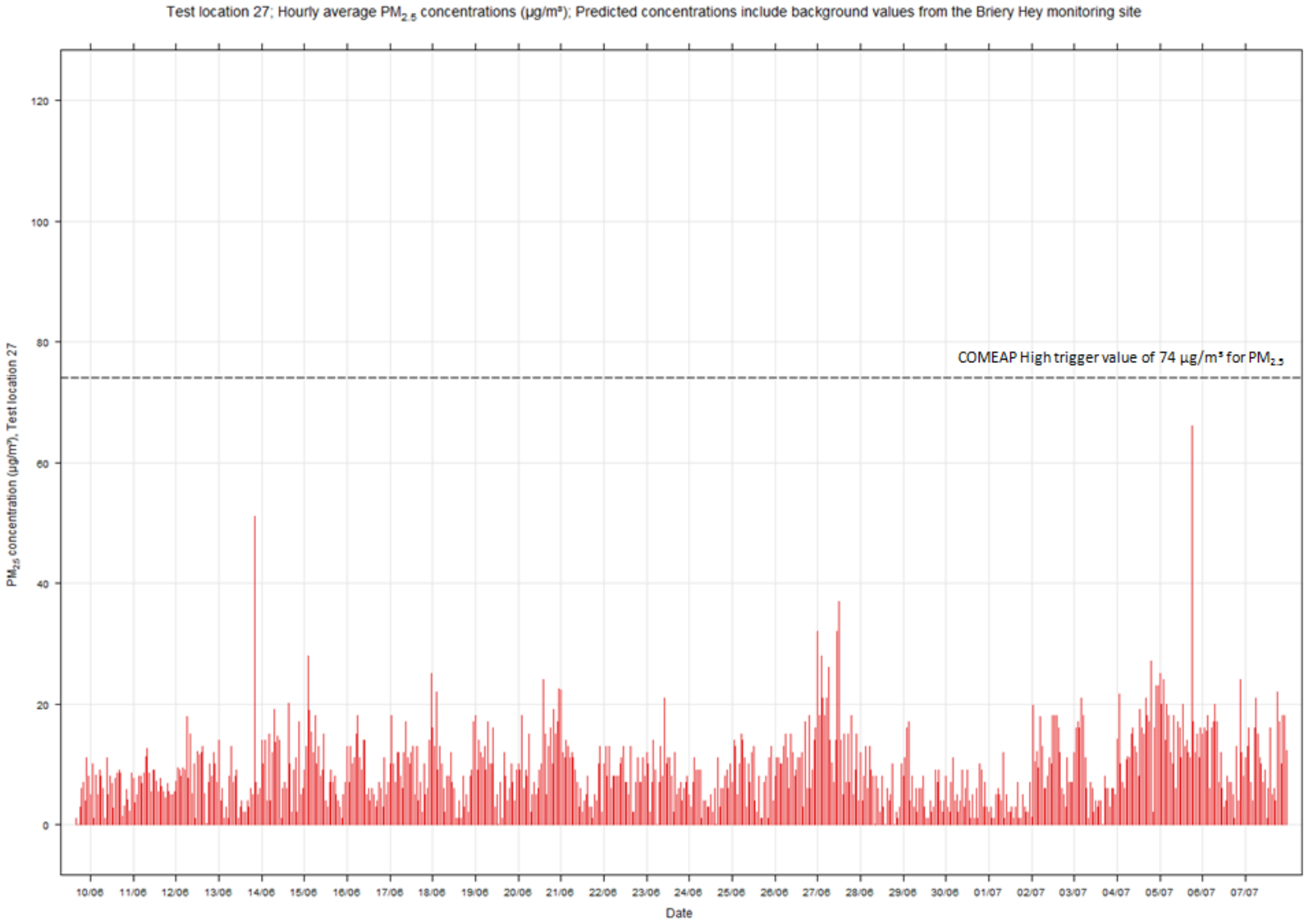

Test location 18W; Hourly average PM2.5 concentrations (µg/m³); Predicted concentrations include background values from the Briery Hey monitoring site
PM2.5 concentration (µg/m³), Test location 18W
120
100
80
60
40
20
0
COMEAP High trigger value of 74 µg/m³ for PM2.5
10/06
11/06
12/06
13/06
14/06
15/06
16/06
17/06
18/06
19/06
20/06
21/06
22/06
23/06
24/06
25/06
26/06
27/06
28/06
29/06
30/06
01/07
02/07
03/07
04/07
05/07
06/07
07/07
Date

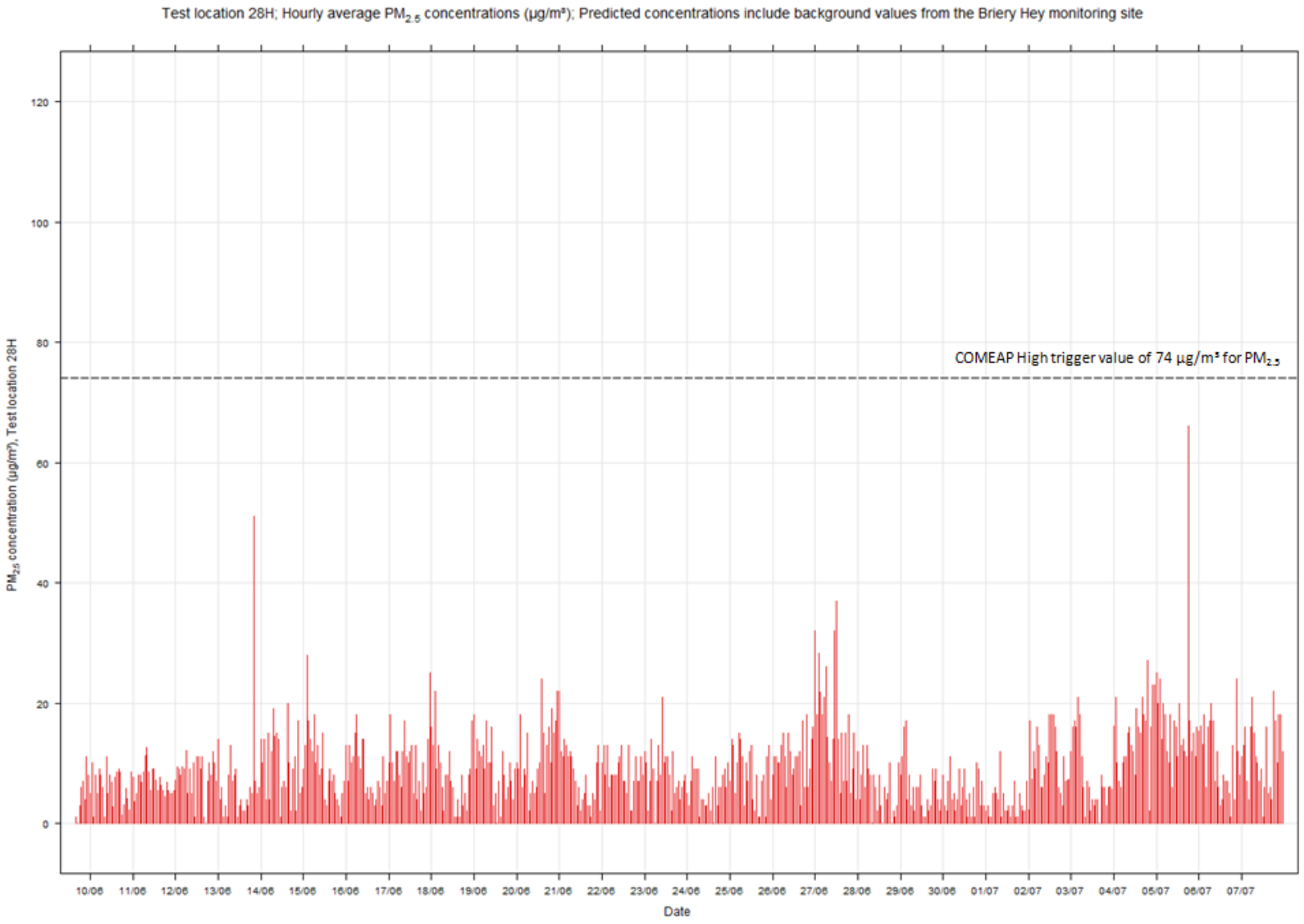

Test location 20; Hourly average PM2.5 concentrations (µg/m³); Predicted concentrations include background values from the Briery Hey monitoring site
PM2.5 concentration (µg/m³), Test location 20
120
100
80
60
40
20
0
COMEAP High trigger value of 74 µg/m³ for PM2.5
10/06
11/06
12/06
13/06
14/06
15/06
16/06
17/06
18/06
19/06
20/06
21/06
22/06
23/06
24/06
25/06
26/06
27/06
28/06
29/06
30/06
01/07
02/07
03/07
04/07
05/07
06/07
07/07
Date

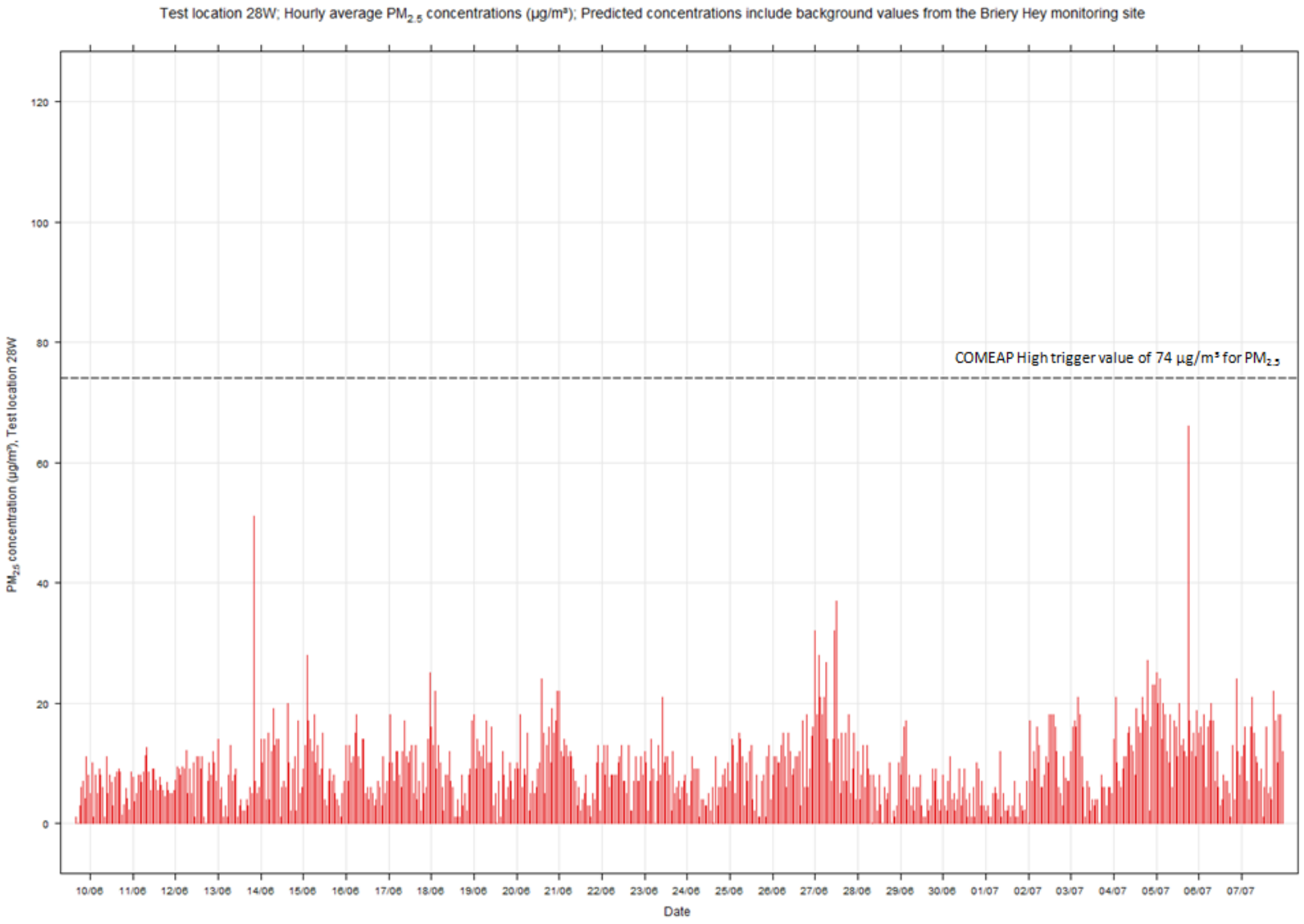

Test location 23; Hourly average PM2.5 concentrations (µg/m³); Predicted concentrations include background values from the Briery Hey monitoring site
PM2.5 concentration (µg/m³), Test location 23
120
100
80
60
40
20
0
COMEAP High trigger value of 74 µg/m³ for PM2.5
10/06
11/06
12/06
13/06
14/06
15/06
16/06
17/06
18/06
19/06
20/06
21/06
22/06
23/06
24/06
25/06
26/06
27/06
28/06
29/06
30/06
01/07
02/07
03/07
04/07
05/07
06/07
07/07
Date

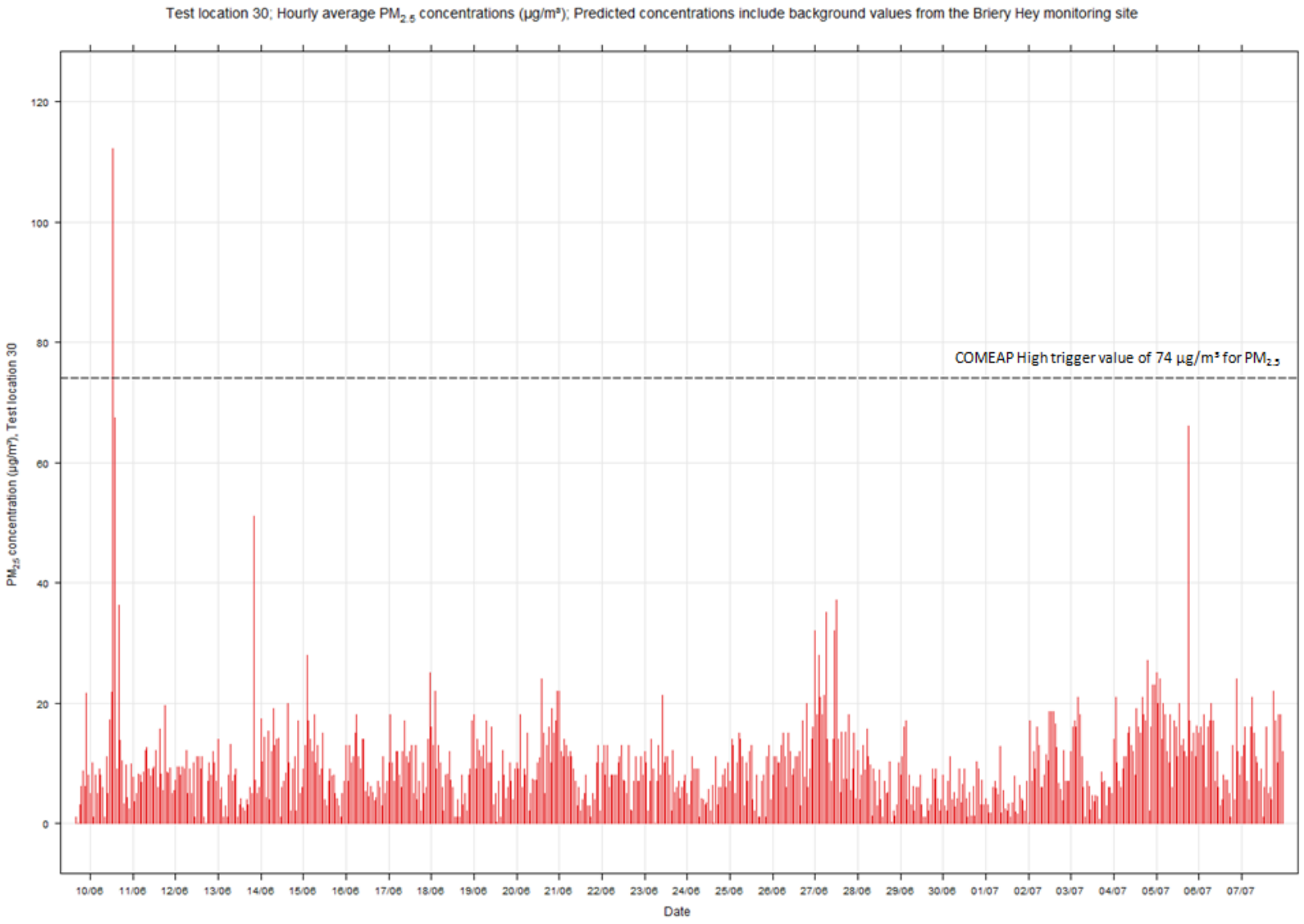

Test location 24; Hourly average PM2.5 concentrations (µg/m³); Predicted concentrations include background values from the Briery Hey monitoring site
PM2.5 concentration (µg/m³), Test location 24
120
100
80
60
40
20
0
COMEAP High trigger value of 74 µg/m³ for PM2.5
10/06
11/06
12/06
13/06
14/06
15/06
16/06
17/06
18/06
19/06
20/06
21/06
22/06
23/06
24/06
25/06
26/06
27/06
28/06
29/06
30/06
01/07
02/07
03/07
04/07
05/07
06/07
07/07
Date

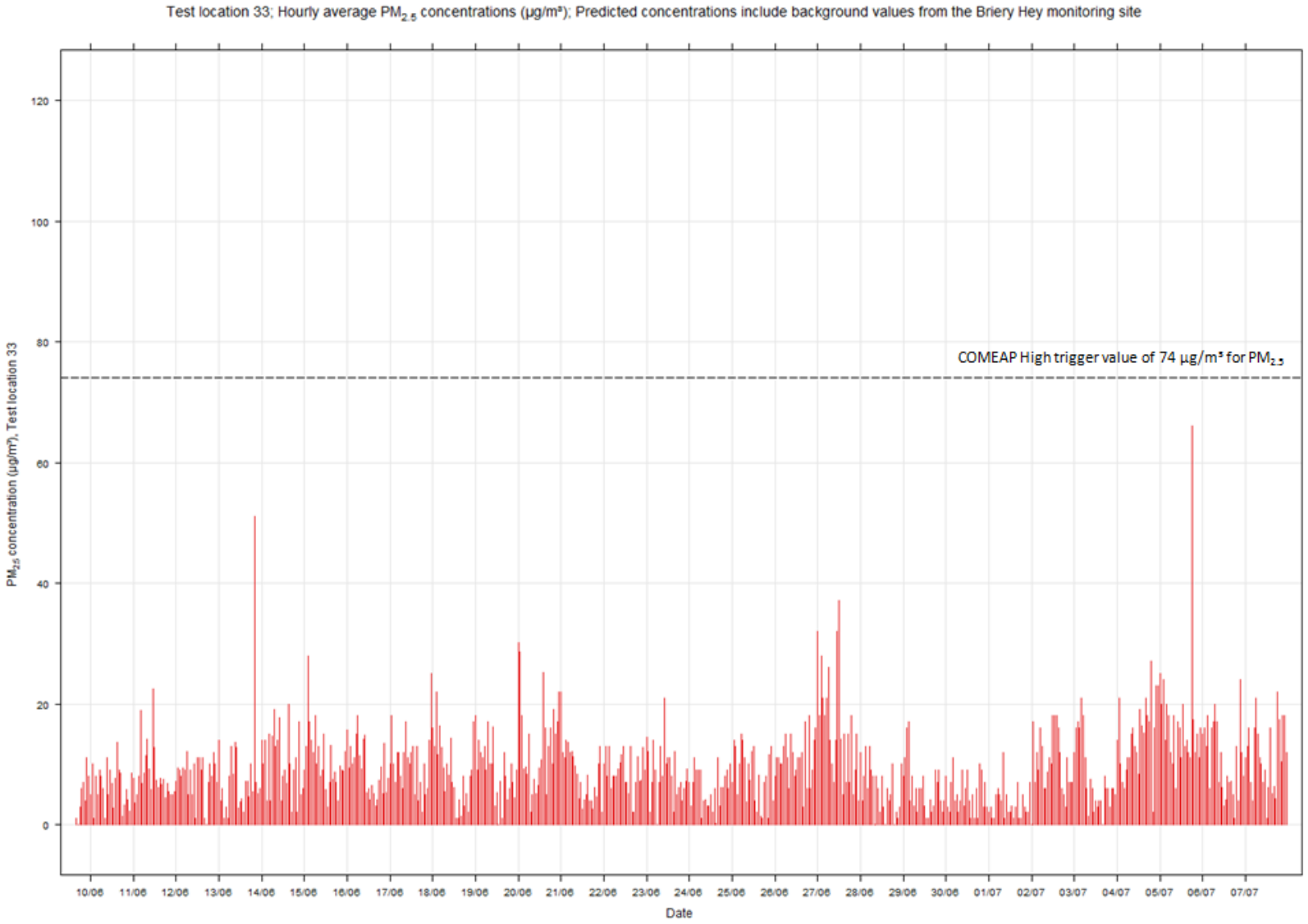

Test location 25; Hourly average PM2.5 concentrations (µg/m³); Predicted concentrations include background values from the Briery Hey monitoring site
COMEAP High trigger value of 74 µg/m³ for PM2.5
PM2.5 concentration (µg/m³), Test location 25
0
20
40
60
80
100
120
10/06
11/06
12/06
13/06
14/06
15/06
16/06
17/06
18/06
19/06
20/06
21/06
22/06
23/06
24/06
25/06
26/06
27/06
28/06
29/06
30/06
01/07
02/07
03/07
04/07
05/07
06/07
07/07
Date

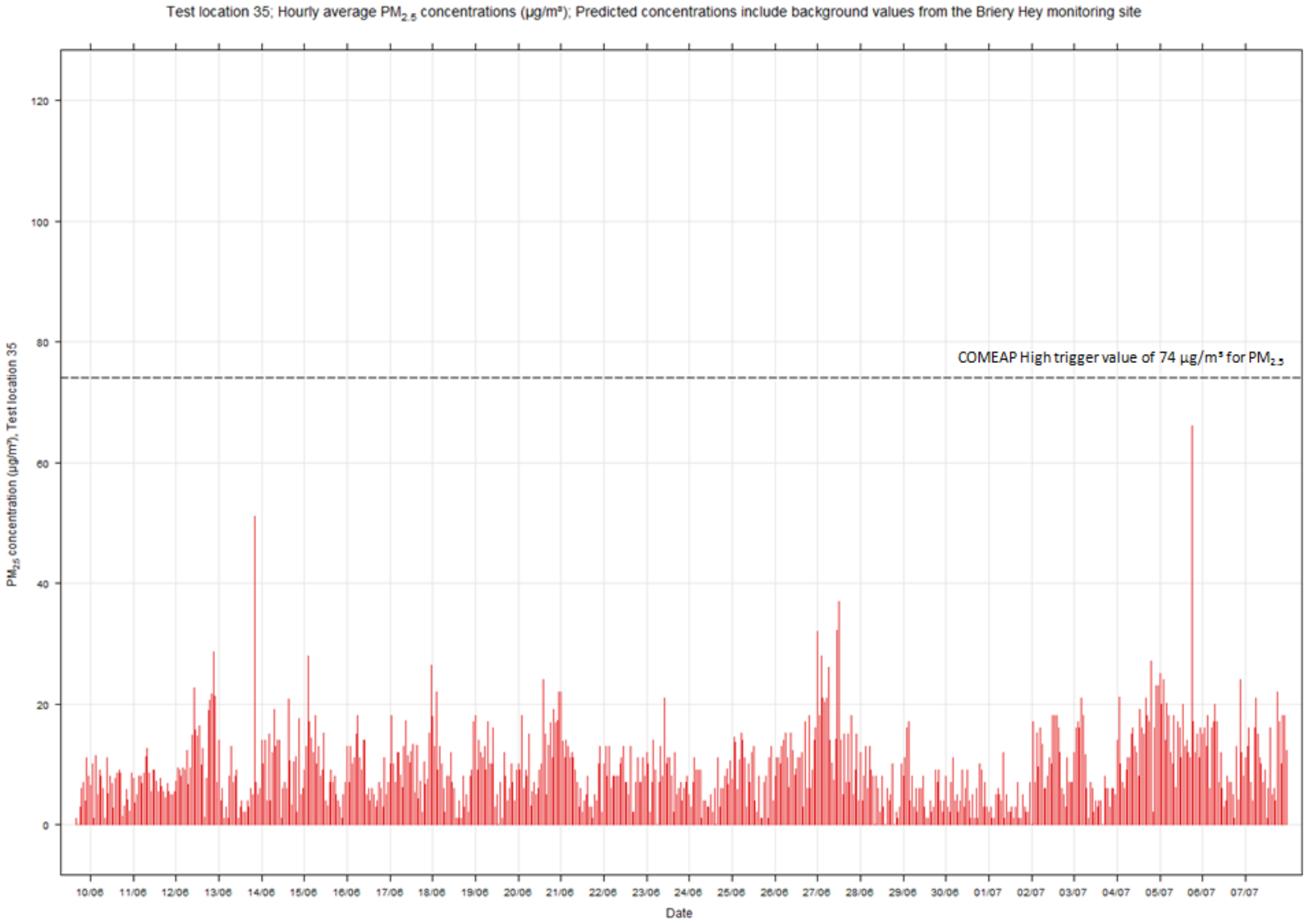

Test location 27; Hourly average PM2.5 concentrations (µg/m³); Predicted concentrations include background values from the Briery Hey monitoring site
PM2.5 concentration (µg/m³), Test location 27
120
100
80
60
40
20
0
COMEAP High trigger value of 74 µg/m³ for PM2.5
10/06
11/06
12/06
13/06
14/06
15/06
16/06
17/06
18/06
19/06
20/06
21/06
22/06
23/06
24/06
25/06
26/06
27/06
28/06
29/06
30/06
01/07
02/07
03/07
04/07
05/07
06/07
07/07
Date


Test location 28H; Hourly average PM2.5 concentrations (µg/m³); Predicted concentrations include background values from the Briery Hey monitoring site
PM2.5 concentration (µg/m³), Test location 28H
120
100
80
60
40
20
0
COMEAP High trigger value of 74 µg/m³ for PM2.5
10/06
11/06
12/06
13/06
14/06
15/06
16/06
17/06
18/06
19/06
20/06
21/06
22/06
23/06
24/06
25/06
26/06
27/06
28/06
29/06
30/06
01/07
02/07
03/07
04/07
05/07
06/07
07/07
Date


Test location 28W; Hourly average PM2.5 concentrations (µg/m³); Predicted concentrations include background values from the Briery Hey monitoring site
PM2.5 concentration (µg/m³), Test location 28W
120
100
80
60
40
20
0
COMEAP High trigger value of 74 µg/m³ for PM2.5
10/06
11/06
12/06
13/06
14/06
15/06
16/06
17/06
18/06
19/06
20/06
21/06
22/06
23/06
24/06
25/06
26/06
27/06
28/06
29/06
30/06
01/07
02/07
03/07
04/07
05/07
06/07
07/07
Date


Test location 30; Hourly average PM2.5 concentrations (µg/m³); Predicted concentrations include background values from the Briery Hey monitoring site
PM2.5 concentration (µg/m³), Test location 30
120
100
80
60
40
20
0
COMEAP High trigger value of 74 µg/m³ for PM2.5
10/06
11/06
12/06
13/06
14/06
15/06
16/06
17/06
18/06
19/06
20/06
21/06
22/06
23/06
24/06
25/06
26/06
27/06
28/06
29/06
30/06
01/07
02/07
03/07
04/07
05/07
06/07
07/07
Date


Test location 33; Hourly average PM2.5 concentrations (µg/m³); Predicted concentrations include background values from the Briery Hey monitoring site
PM2.5 concentration (µg/m³), Test location 33
120
100
80
60
40
20
0
COMEAP High trigger value of 74 µg/m³ for PM2.5
10/06
11/06
12/06
13/06
14/06
15/06
16/06
17/06
18/06
19/06
20/06
21/06
22/06
23/06
24/06
25/06
26/06
27/06
28/06
29/06
30/06
01/07
02/07
03/07
04/07
05/07
06/07
07/07
Date


Test location 35; Hourly average PM2.5 concentrations (µg/m³); Predicted concentrations include background values from the Briery Hey monitoring site
PM2.5 concentration (µg/m³), Test location 35
120
100
80
60
40
20
0
COMEAP High trigger value of 74 µg/m³ for PM2.5
10/06
11/06
12/06
13/06
14/06
15/06
16/06
17/06
18/06
19/06
20/06
21/06
22/06
23/06
24/06
25/06
26/06
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30/06
01/07
02/07
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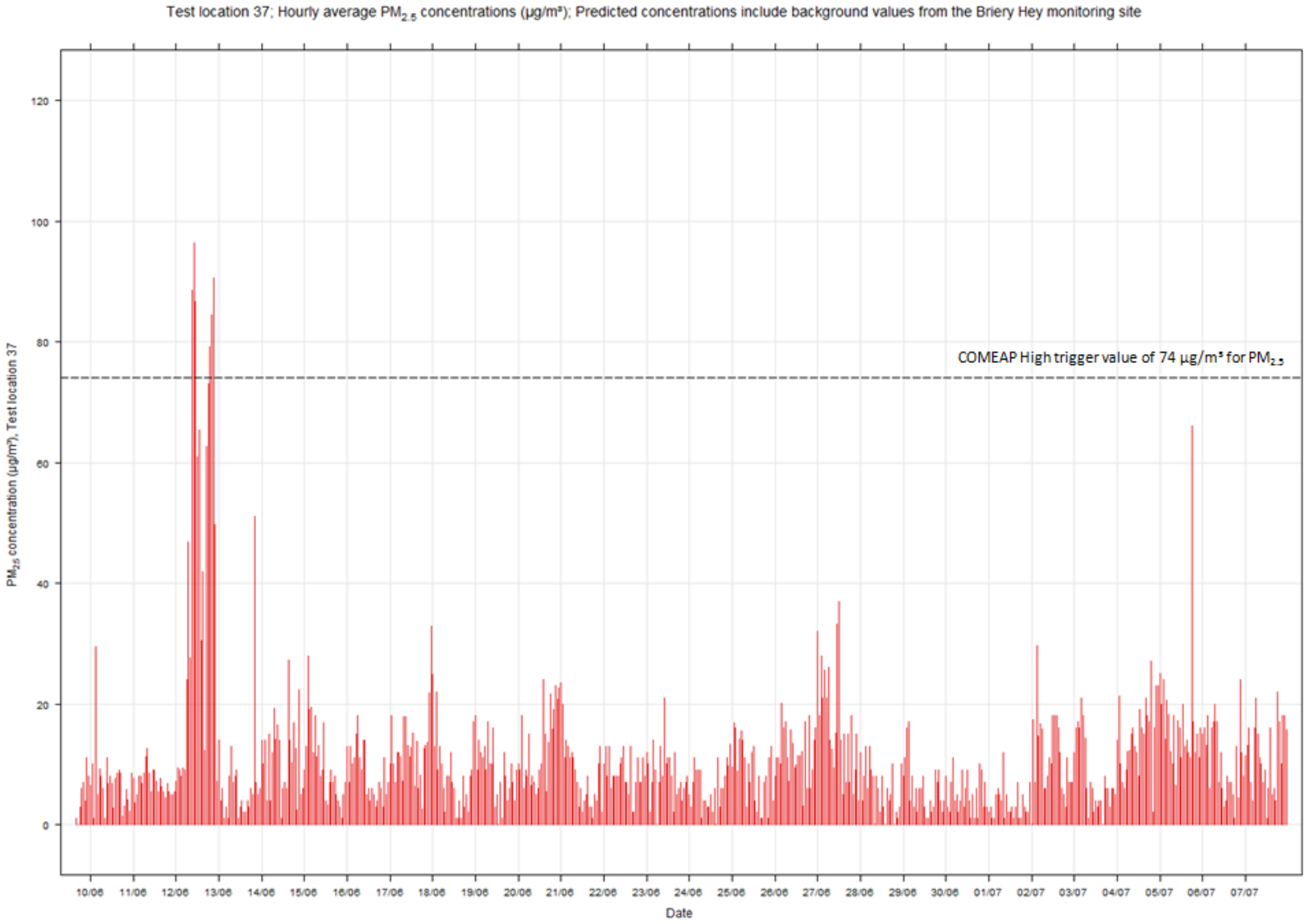
04/07
05/07
06/07
07/07
Date


Test location 37; Hourly average PM2.5 concentrations (µg/m³); Predicted concentrations include background values from the Briery Hey monitoring site
COMEAP High trigger value of 74 µg/m³ for PM2.5
PM2.5 concentration (µg/m³), Test location 37
0
20
40
60
80
100
120
10/06
11/06
12/06
13/06
14/06
15/06
16/06
17/06
18/06
19/06
20/06
21/06
22/06
23/06
24/06
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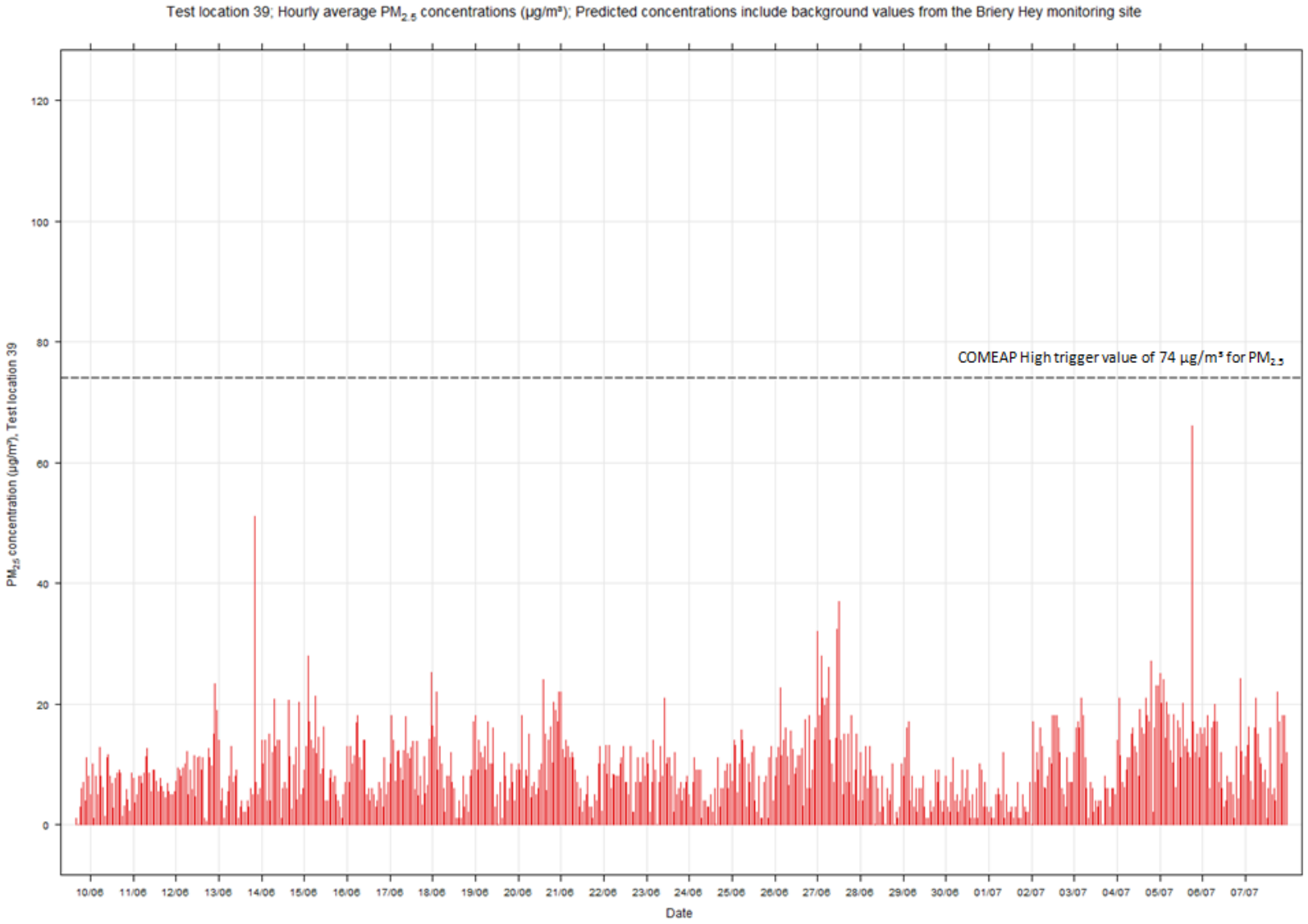
03/07
04/07
05/07
06/07
07/07
Date


Test location 39; Hourly average PM2.5 concentrations (µg/m³); Predicted concentrations include background values from the Briery Hey monitoring site
PM2.5 concentration (µg/m³), Test location 39
120
100
80
60
40
20
0
COMEAP High trigger value of 74 µg/m³ for PM2.5
10/06
11/06
12/06
13/06
14/06
15/06
16/06
17/06
18/06
19/06
20/06
21/06
22/06
23/06
24/06
25/06
26/06
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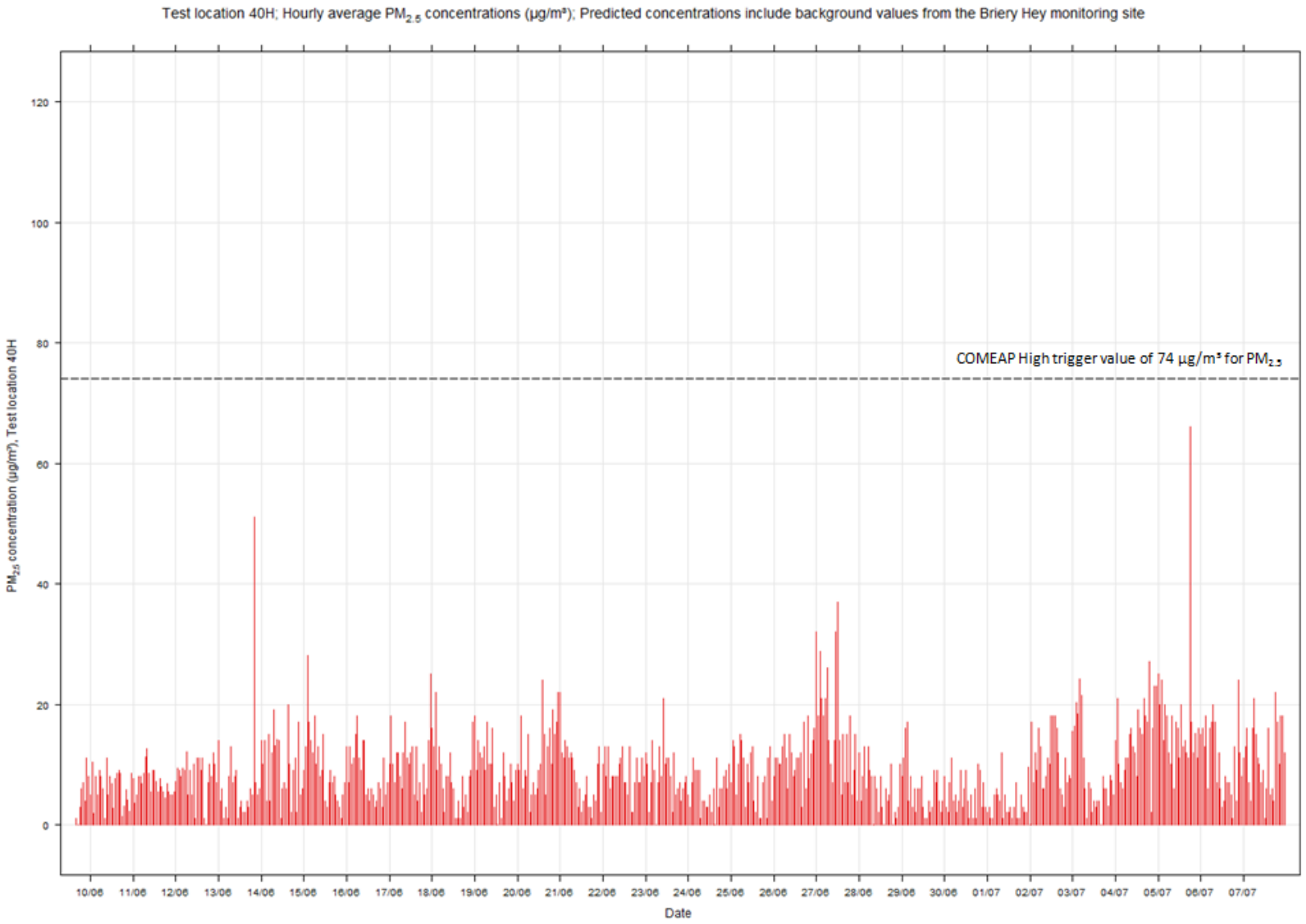
28/06
29/06
30/06
01/07
02/07
03/07
04/07
05/07
06/07
07/07
Date


Test location 40H; Hourly average PM2.5 concentrations (µg/m³); Predicted concentrations include background values from the Briery Hey monitoring site
PM2.5 concentration (µg/m³), Test location 40H
120
100
80
60
40
20
0
COMEAP High trigger value of 74 µg/m³ for PM2.5
10/06
11/06
12/06
13/06
14/06
15/06
16/06
17/06
18/06
19/06
20/06
21/06
22/06
23/06
24/06
25/06
26/06
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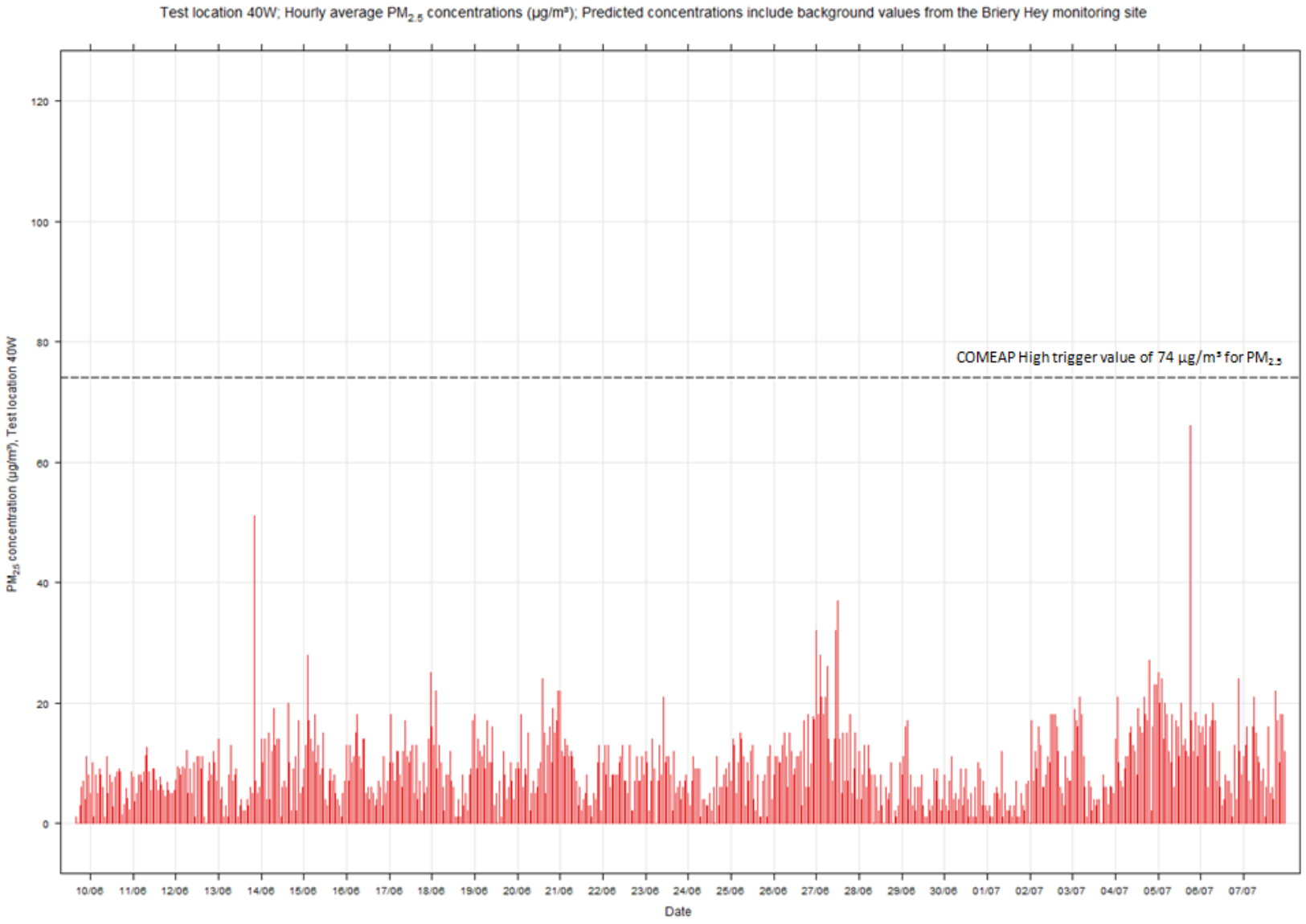
28/06
29/06
30/06
01/07
02/07
03/07
04/07
05/07
06/07
07/07
Date


Test location 40W; Hourly average PM2.5 concentrations (µg/m³); Predicted concentrations include background values from the Briery Hey monitoring site
PM2.5 concentration (µg/m³), Test location 40W
120
100
80
60
40
20
0
COMEAP High trigger value of 74 µg/m³ for PM2.5
10/06
11/06
12/06
13/06
14/06
15/06
16/06
17/06
18/06
19/06
20/06
21/06
22/06
23/06
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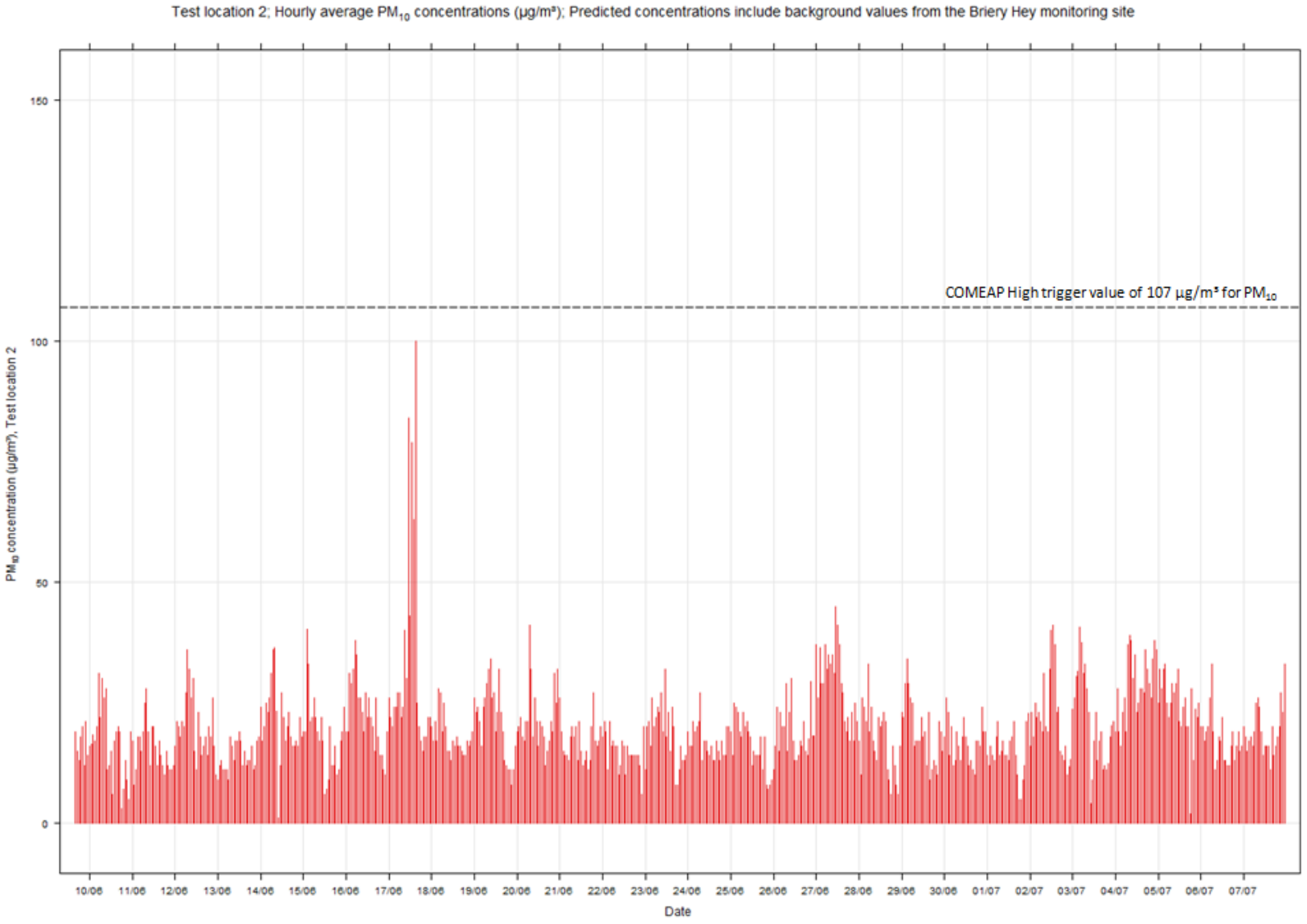
Date

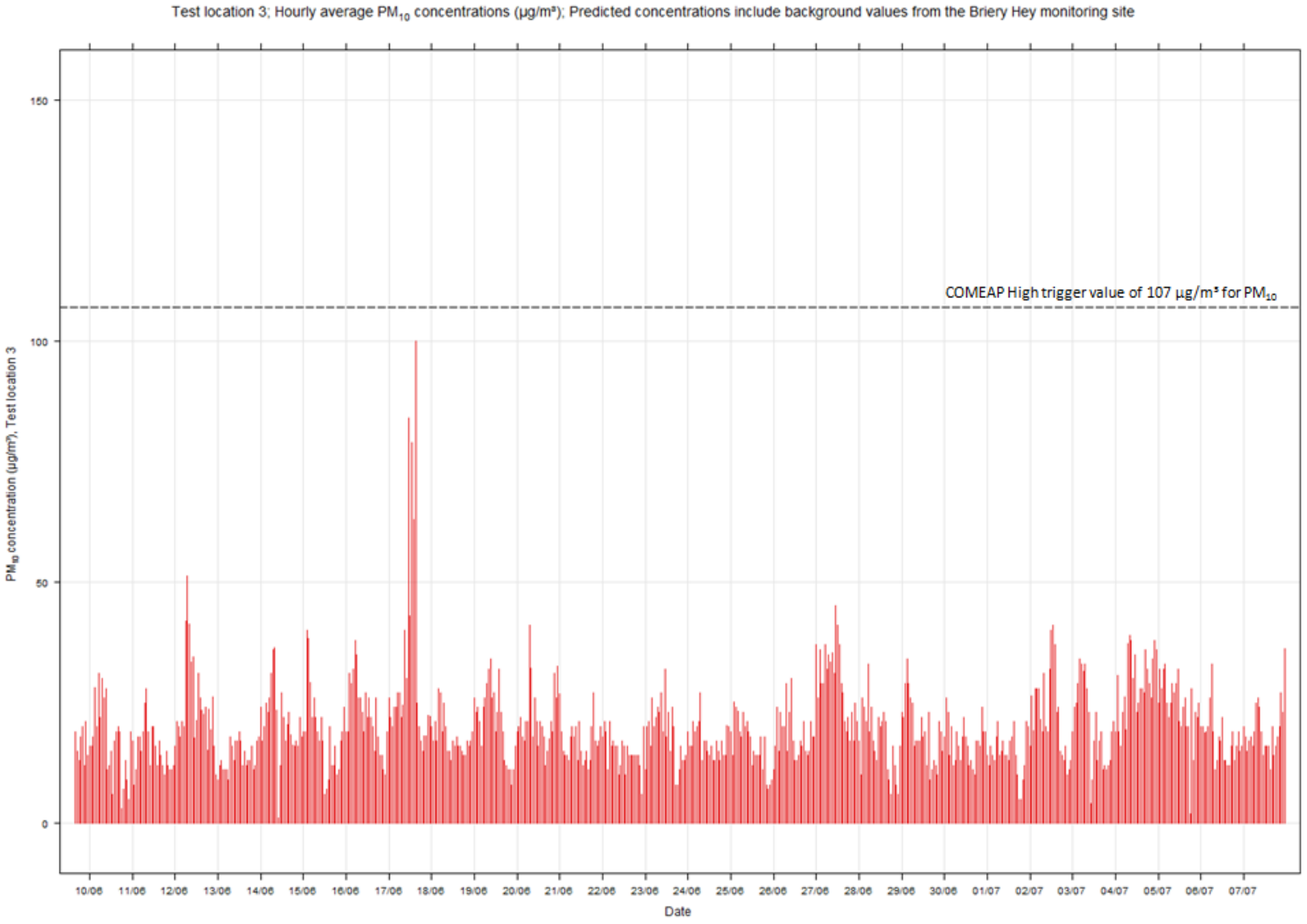
Sonae GLO: Further Modelling

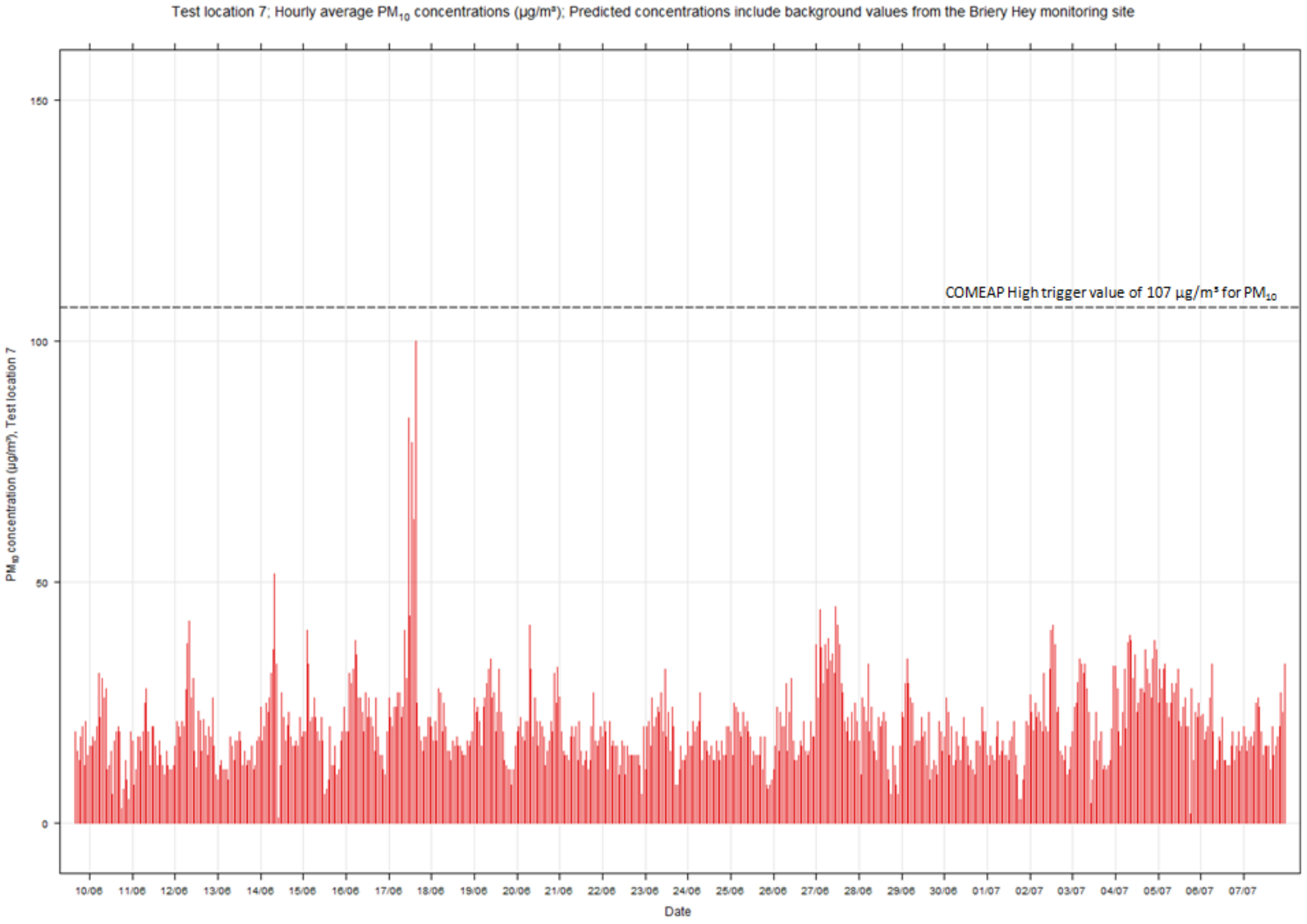
6. Histograms of modelled hourly concentrations for the 20 Test Claimants

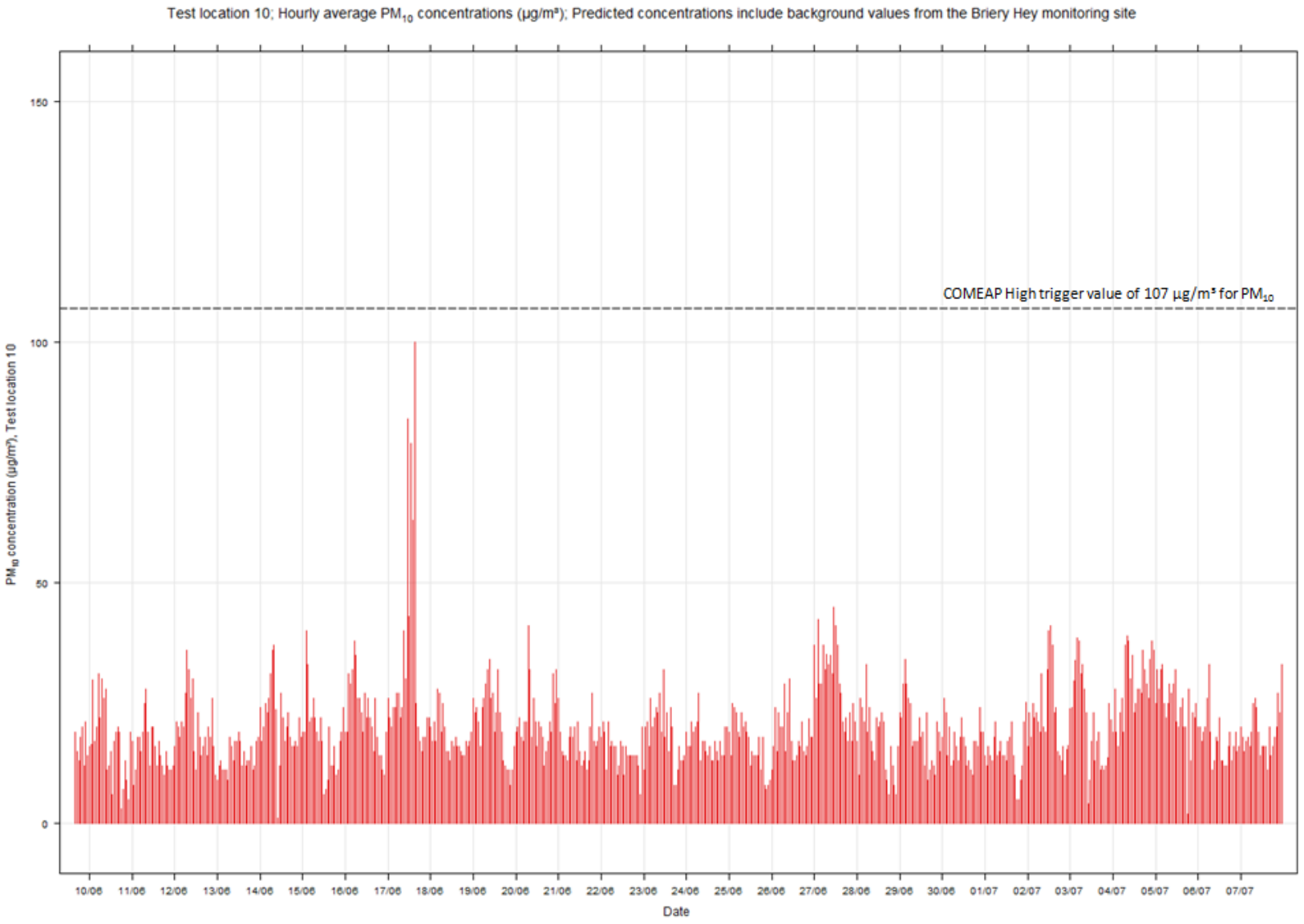
Modelled PM_{10} concentrations compared against the COMEAP High trigger value of 107 $\mu g/m^3$

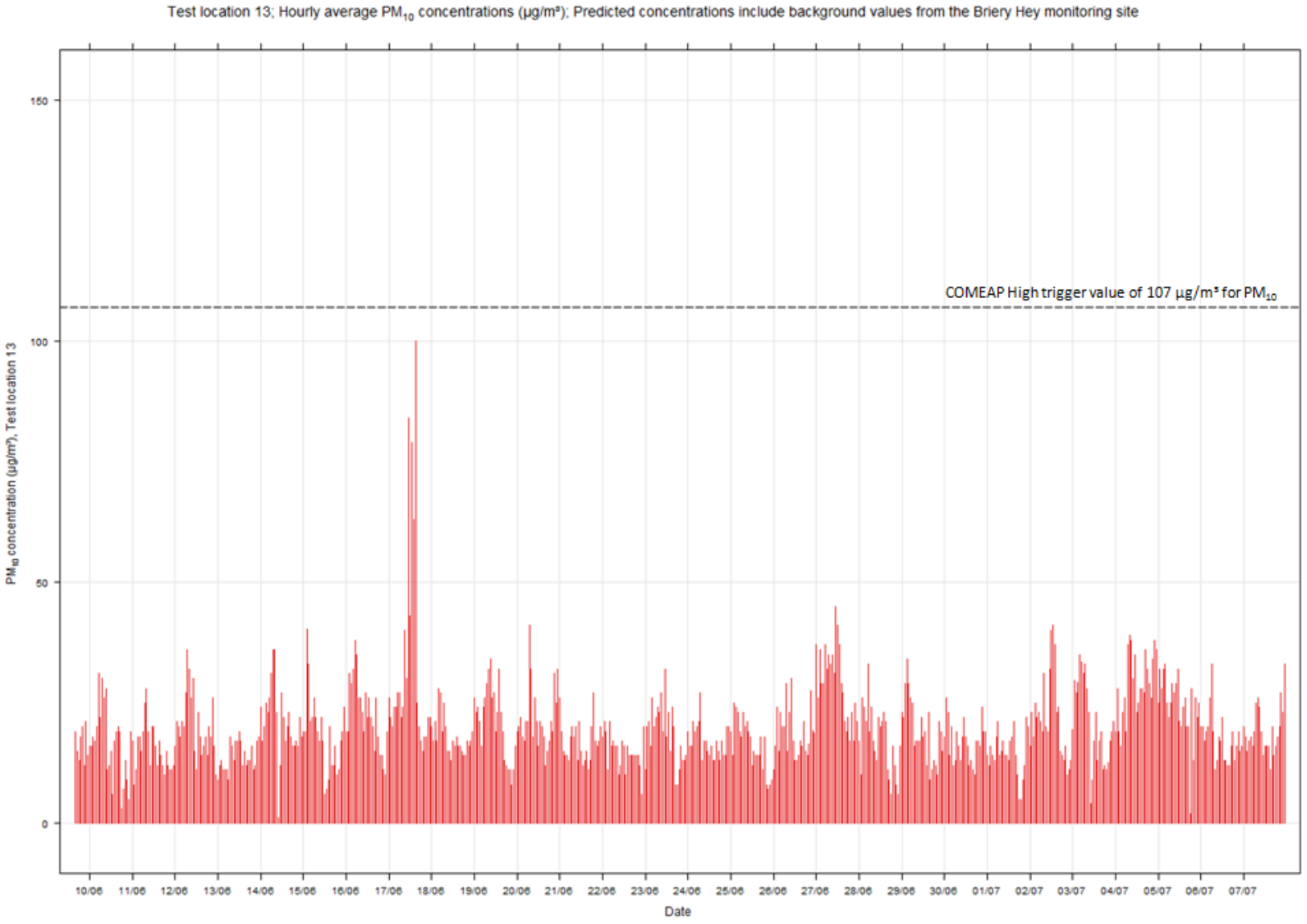
The 23 time series histograms in this document show modelled hourly average concentrations of PM_{10} at the locations of the 20 Test Claimants. Modelled concentrations include the modelled contribution from the fire and background concentrations using measurements from the Briery Hey monitoring site.

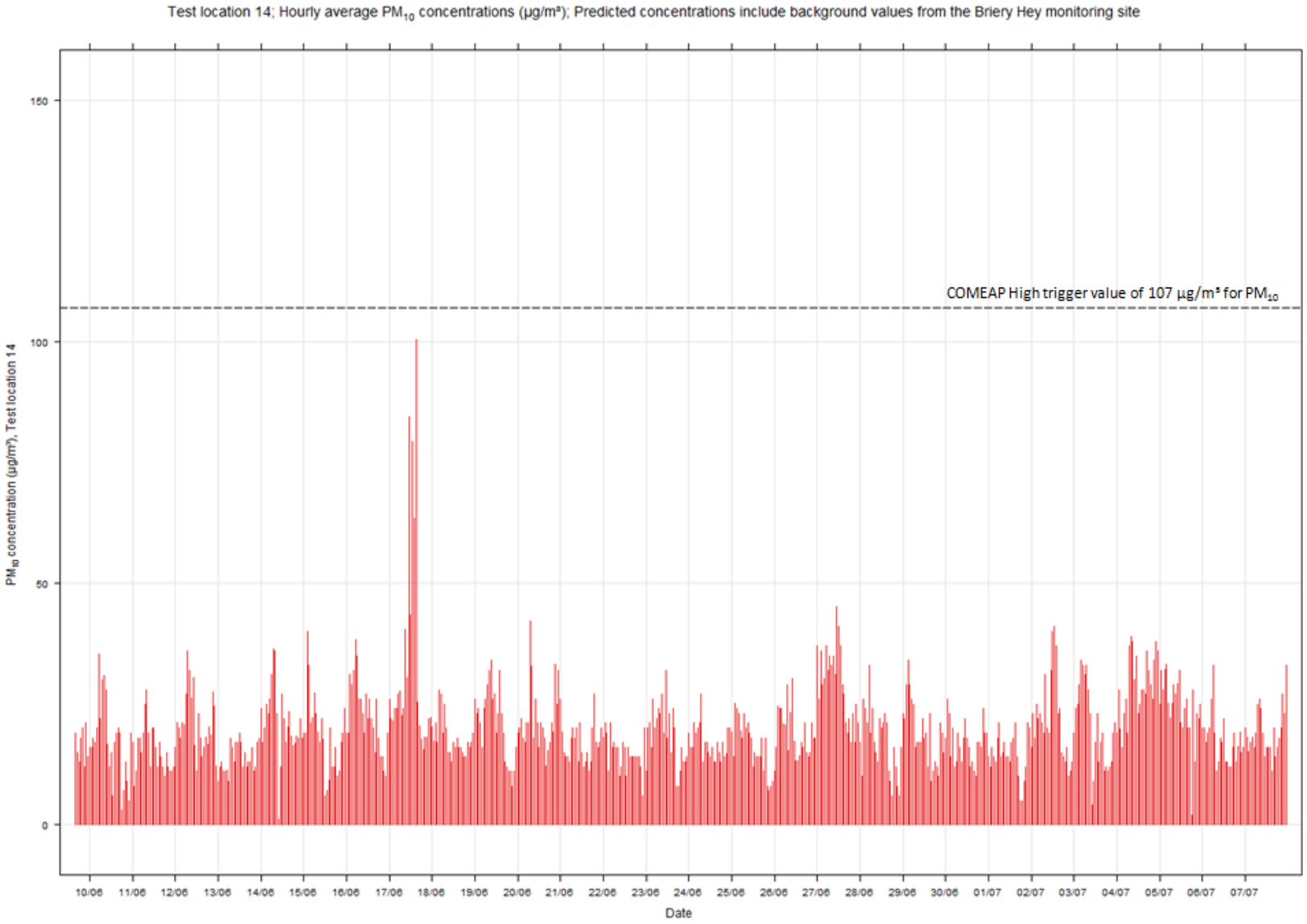

Test location 2; Hourly average PM10 concentrations (µg/m³); Predicted concentrations include background values from the Briery Hey monitoring site
COMEAP High trigger value of 107 µg/m³ for PM10
PM10 concentration (µg/m³), Test location 2
150
100
50
0
10/06
11/06
12/06
13/06
14/06
15/06
16/06
17/06
18/06
19/06
20/06
21/06
22/06
23/06
24/06
25/06
26/06
27/06
28/06
29/06
30/06
01/07
02/07
03/07
04/07
05/07
06/07
07/07
Date

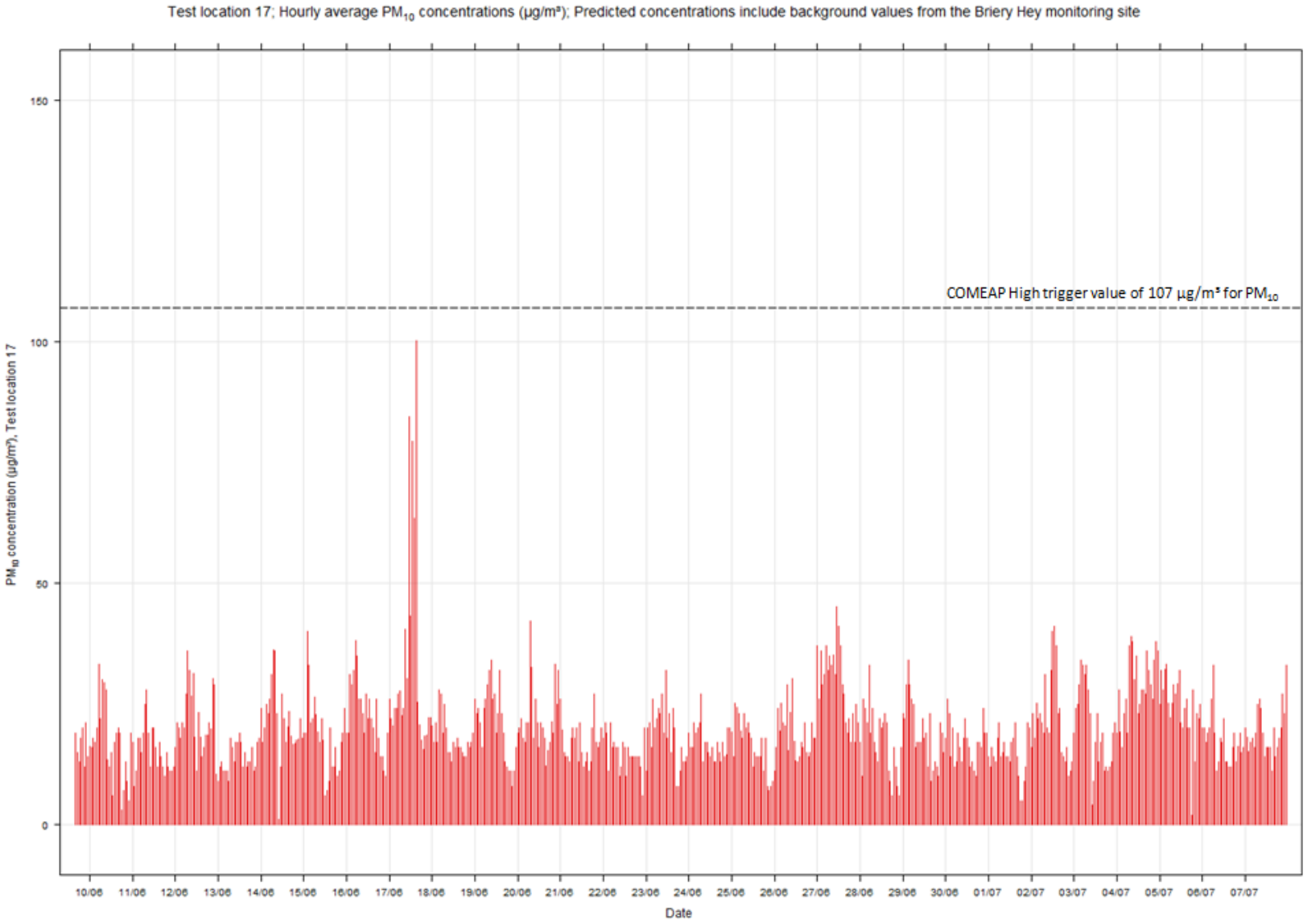

Test location 3; Hourly average PM10 concentrations (µg/m³); Predicted concentrations include background values from the Briery Hey monitoring site
COMEAP High trigger value of 107 µg/m³ for PM10
PM10 concentration (µg/m³), Test location 3
150
100
50
0
10/06
11/06
12/06
13/06
14/06
15/06
16/06
17/06
18/06
19/06
20/06
21/06
22/06
23/06
24/06
25/06
26/06
27/06
28/06
29/06
30/06
01/07
02/07
03/07
04/07
05/07
06/07
07/07
Date

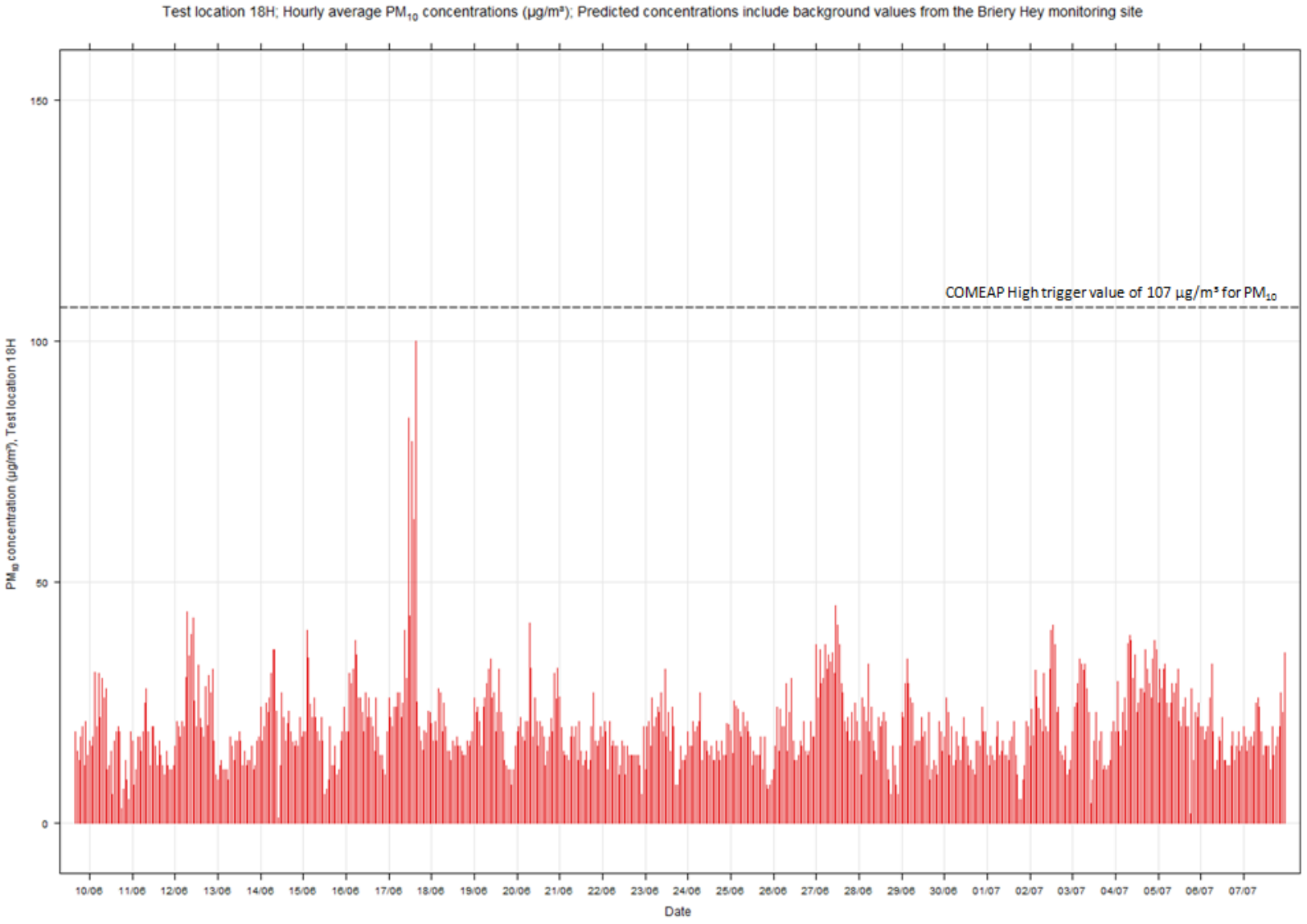

Test location 7; Hourly average PM10 concentrations (µg/m³); Predicted concentrations include background values from the Briery Hey monitoring site
COMEAP High trigger value of 107 µg/m³ for PM10
PM10 concentration (µg/m³), Test location 7
150
100
50
0
10/06
11/06
12/06
13/06
14/06
15/06
16/06
17/06
18/06
19/06
20/06
21/06
22/06
23/06
24/06
25/06
26/06
27/06
28/06
29/06
30/06
01/07
02/07
03/07
04/07
05/07
06/07
07/07
Date

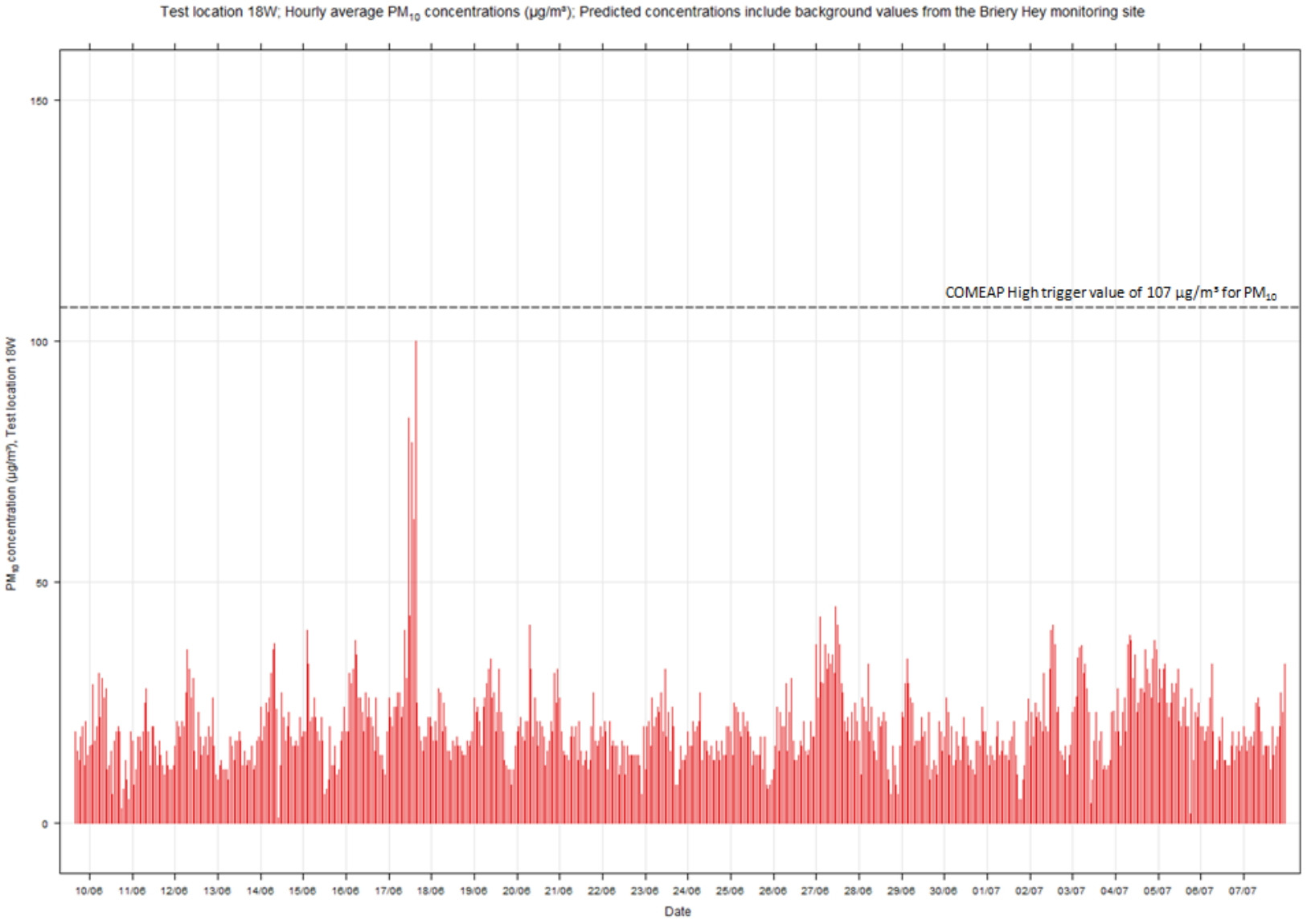

Test location 10; Hourly average PM10 concentrations (µg/m³); Predicted concentrations include background values from the Briery Hey monitoring site
COMEAP High trigger value of 107 µg/m³ for PM10
PM10 concentration (µg/m³), Test location 10
150
100
50
0
10/06
11/06
12/06
13/06
14/06
15/06
16/06
17/06
18/06
19/06
20/06
21/06
22/06
23/06
24/06
25/06
26/06
27/06
28/06
29/06
30/06
01/07
02/07
03/07
04/07
05/07
06/07
07/07
Date

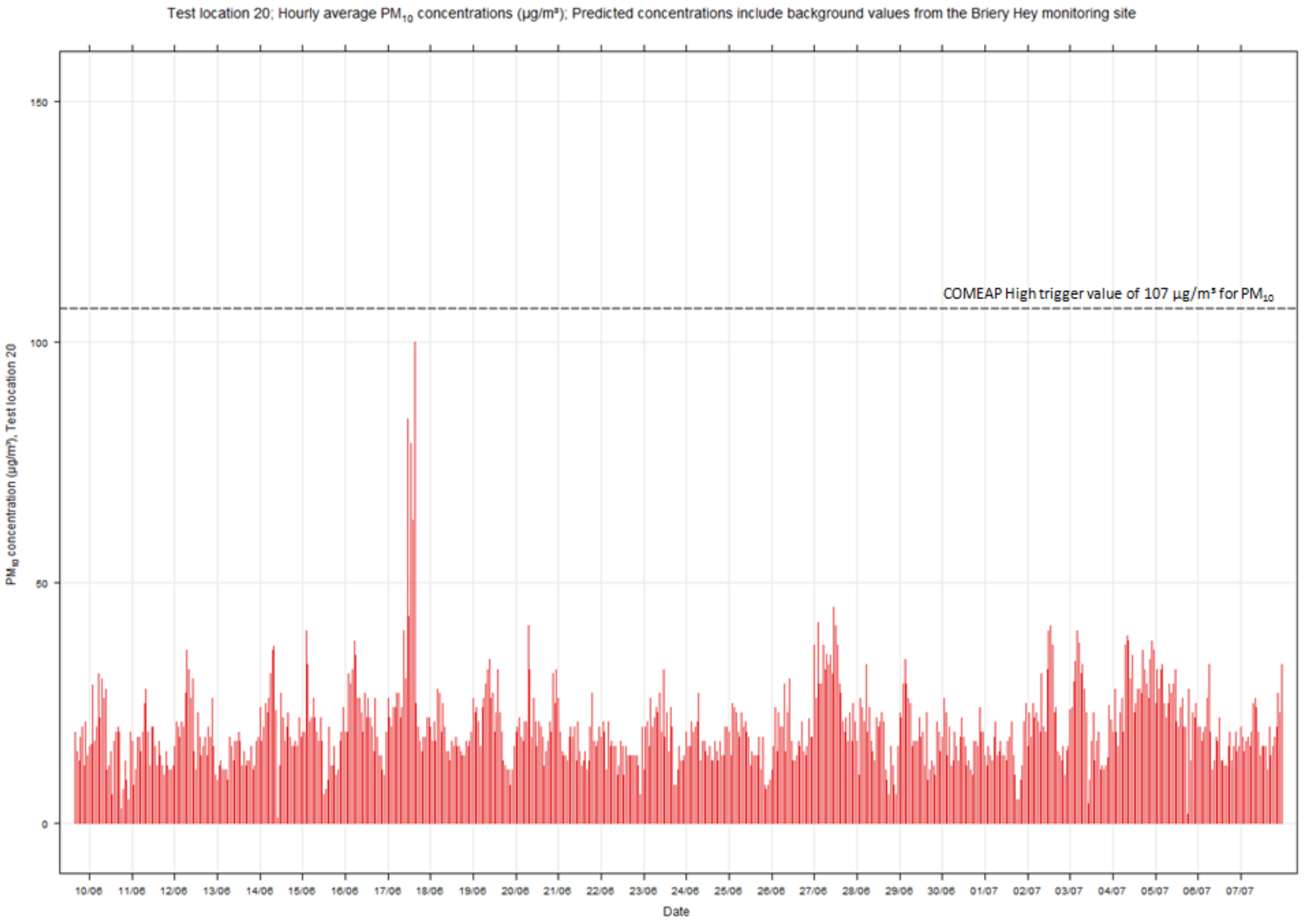

Test location 13; Hourly average PM10 concentrations (µg/m³); Predicted concentrations include background values from the Briery Hey monitoring site
COMEAP High trigger value of 107 µg/m³ for PM10
PM10 concentration (µg/m³), Test location 13
150
100
50
0
10/06
11/06
12/06
13/06
14/06
15/06
16/06
17/06
18/06
19/06
20/06
21/06
22/06
23/06
24/06
25/06
26/06
27/06
28/06
29/06
30/06
01/07
02/07
03/07
04/07
05/07
06/07
07/07
Date

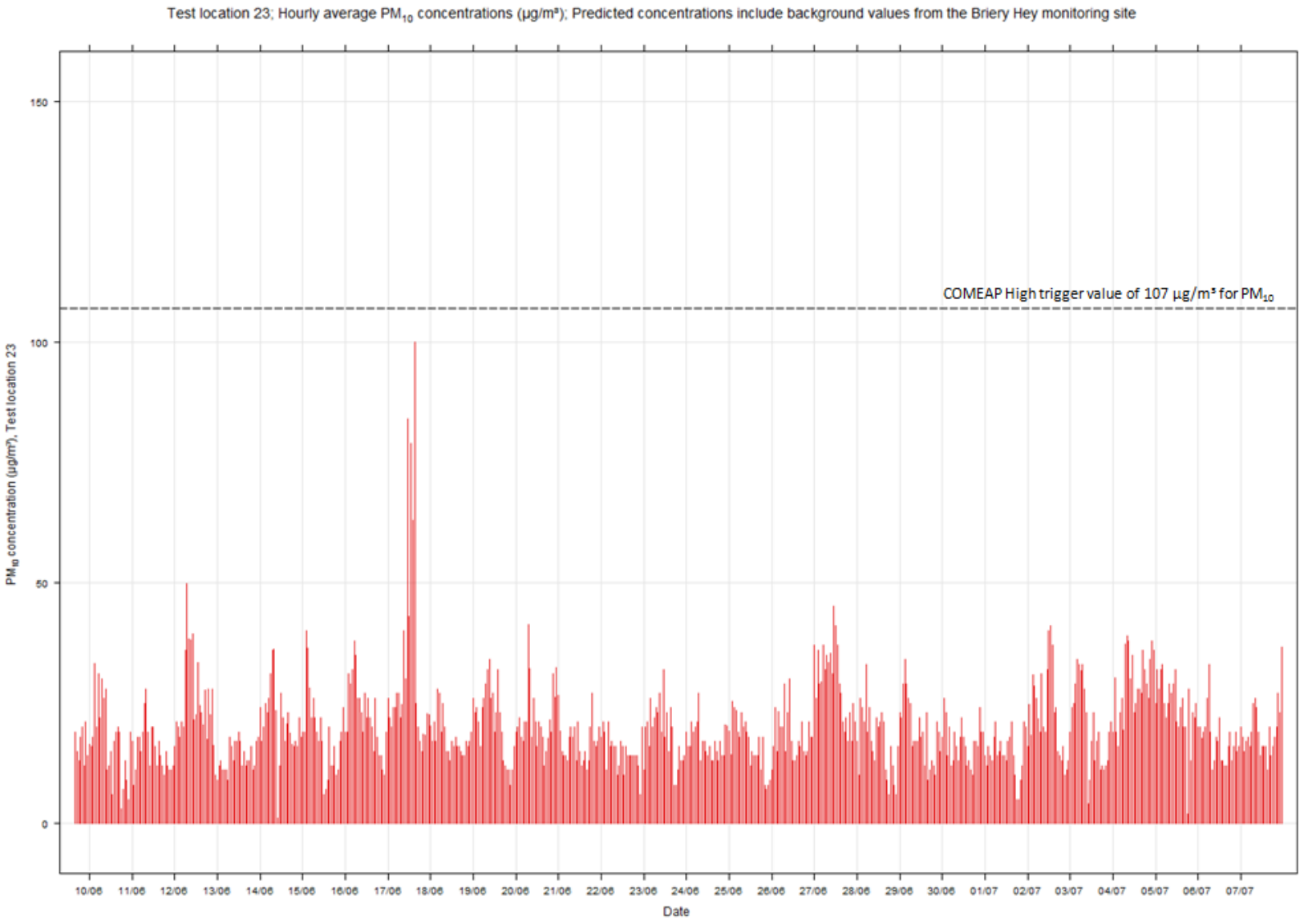

Test location 14; Hourly average PM10 concentrations (µg/m³); Predicted concentrations include background values from the Briery Hey monitoring site
COMEAP High trigger value of 107 µg/m³ for PM10
PM10 concentration (µg/m³), Test location 14
150
100
50
0
10/06
11/06
12/06
13/06
14/06
15/06
16/06
17/06
18/06
19/06
20/06
21/06
22/06
23/06
24/06
25/06
26/06
27/06
28/06
29/06
30/06
01/07
02/07
03/07
04/07
05/07
06/07
07/07
Date

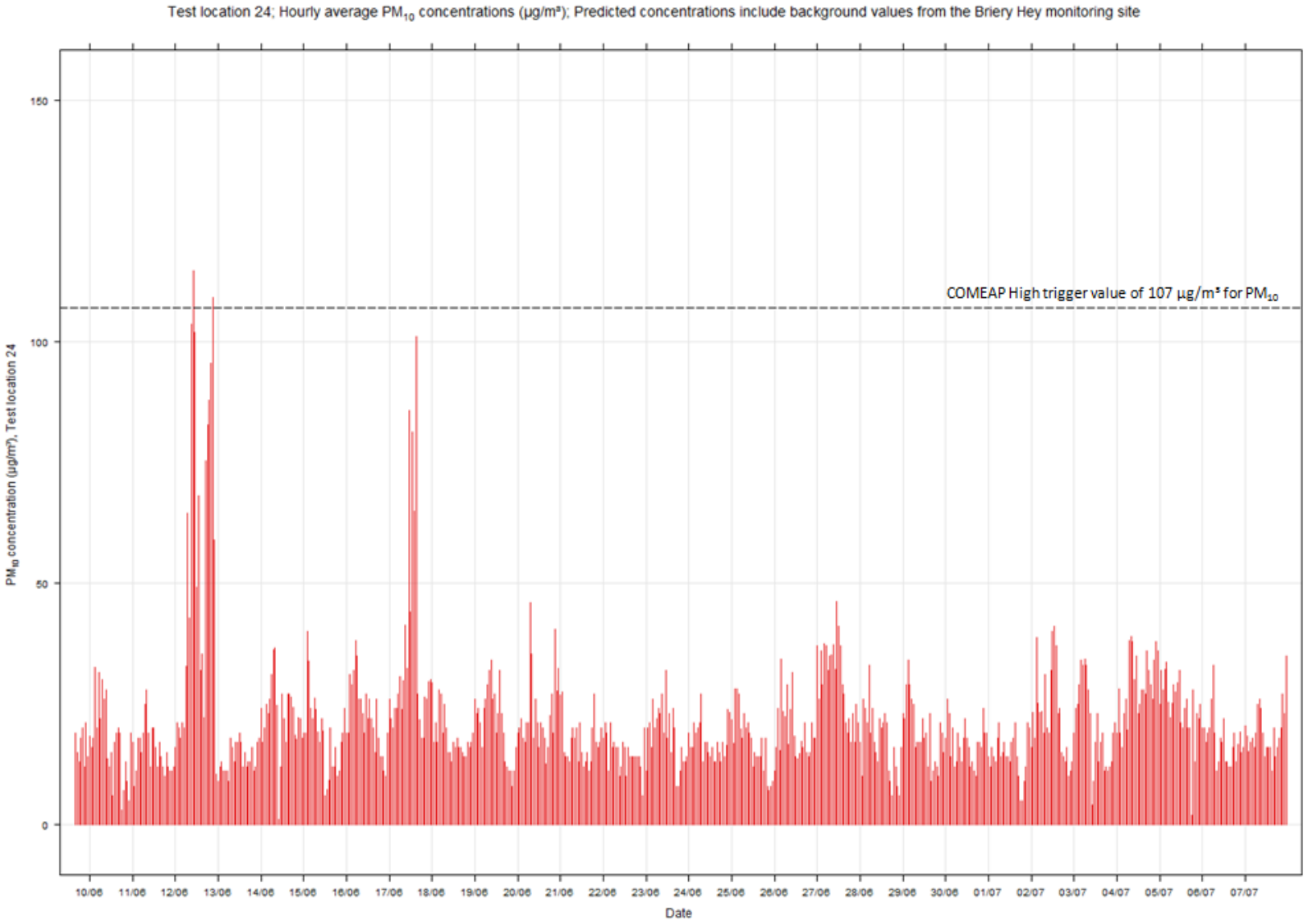

Test location 17; Hourly average PM10 concentrations (µg/m³); Predicted concentrations include background values from the Briery Hey monitoring site
COMEAP High trigger value of 107 µg/m³ for PM10
PM10 concentration (µg/m³), Test location 17
150
100
50
0
10/06
11/06
12/06
13/06
14/06
15/06
16/06
17/06
18/06
19/06
20/06
21/06
22/06
23/06
24/06
25/06
26/06
27/06
28/06
29/06
30/06
01/07
02/07
03/07
04/07
05/07
06/07
07/07
Date

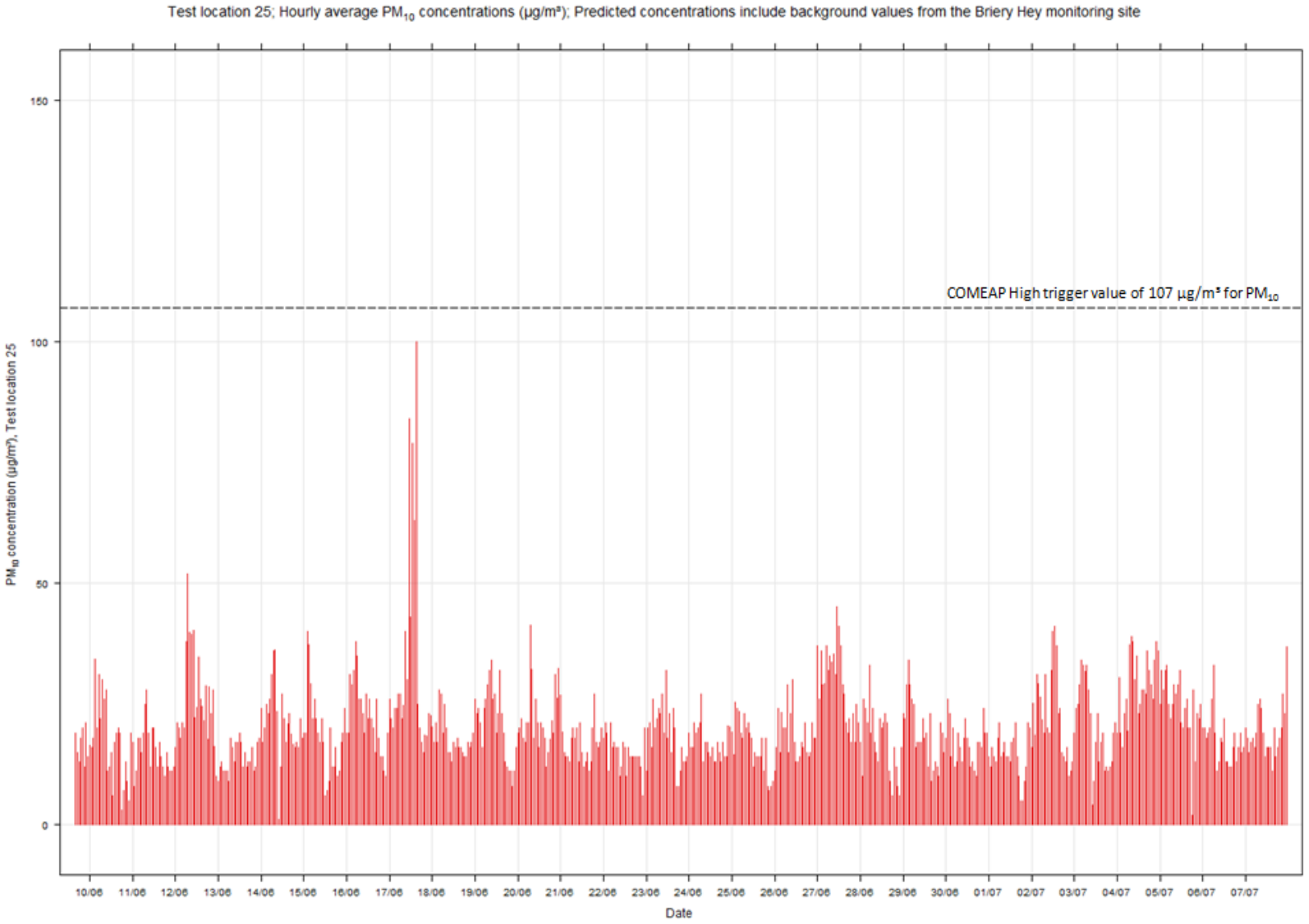

Test location 18H; Hourly average PM10 concentrations (µg/m³); Predicted concentrations include background values from the Briery Hey monitoring site
COMEAP High trigger value of 107 µg/m³ for PM10
PM10 concentration (µg/m³), Test location 18H
150
100
50
0
10/06
11/06
12/06
13/06
14/06
15/06
16/06
17/06
18/06
19/06
20/06
21/06
22/06
23/06
24/06
25/06
26/06
27/06
28/06
29/06
30/06
01/07
02/07
03/07
04/07
05/07
06/07
07/07
Date

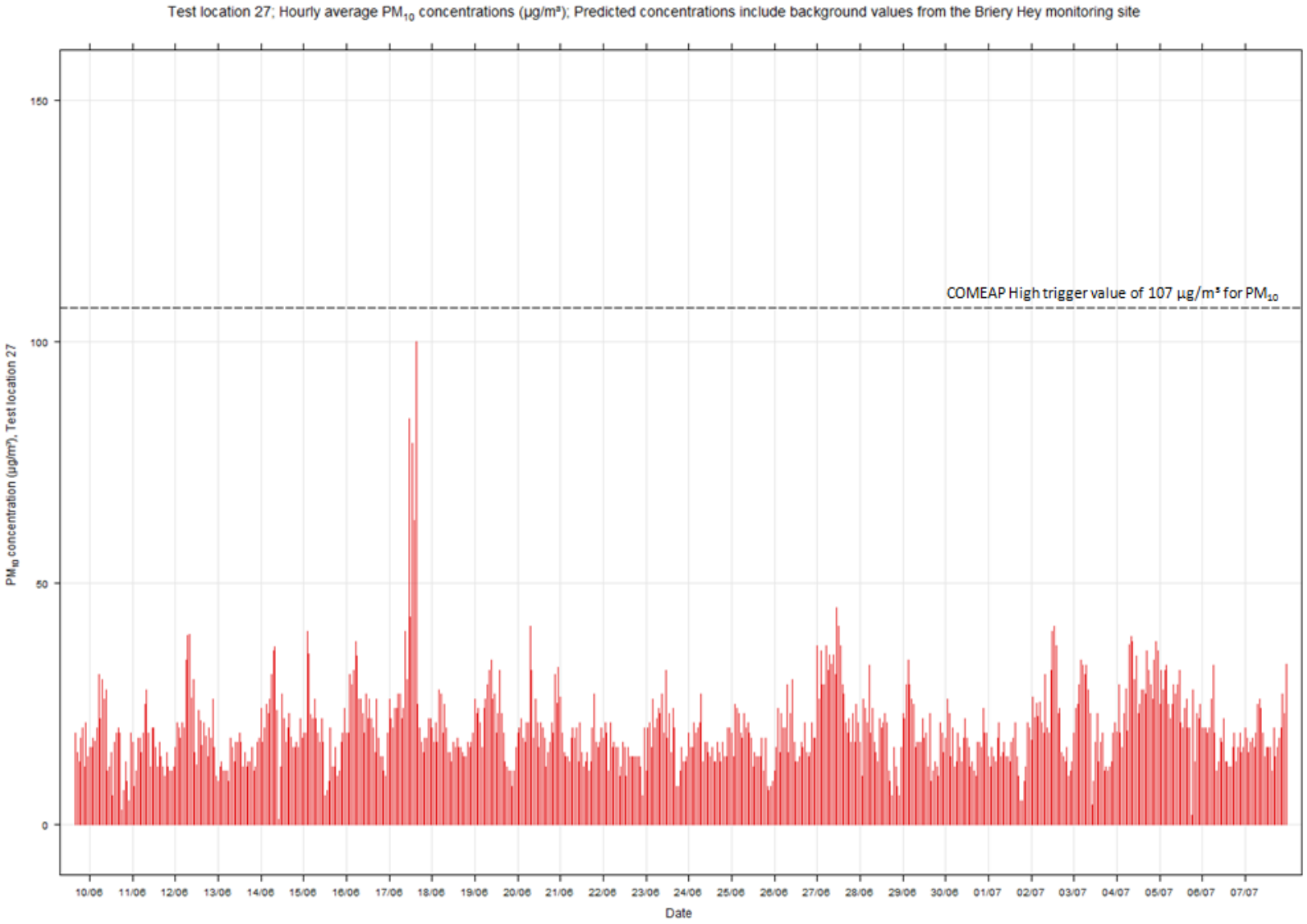

Test location 18W; Hourly average PM10 concentrations (µg/m³); Predicted concentrations include background values from the Briery Hey monitoring site
COMEAP High trigger value of 107 µg/m³ for PM10
PM10 concentration (µg/m³), Test location 18W
150
100
50
0
10/06
11/06
12/06
13/06
14/06
15/06
16/06
17/06
18/06
19/06
20/06
21/06
22/06
23/06
24/06
25/06
26/06
27/06
28/06
29/06
30/06
01/07
02/07
03/07
04/07
05/07
06/07
07/07
Date

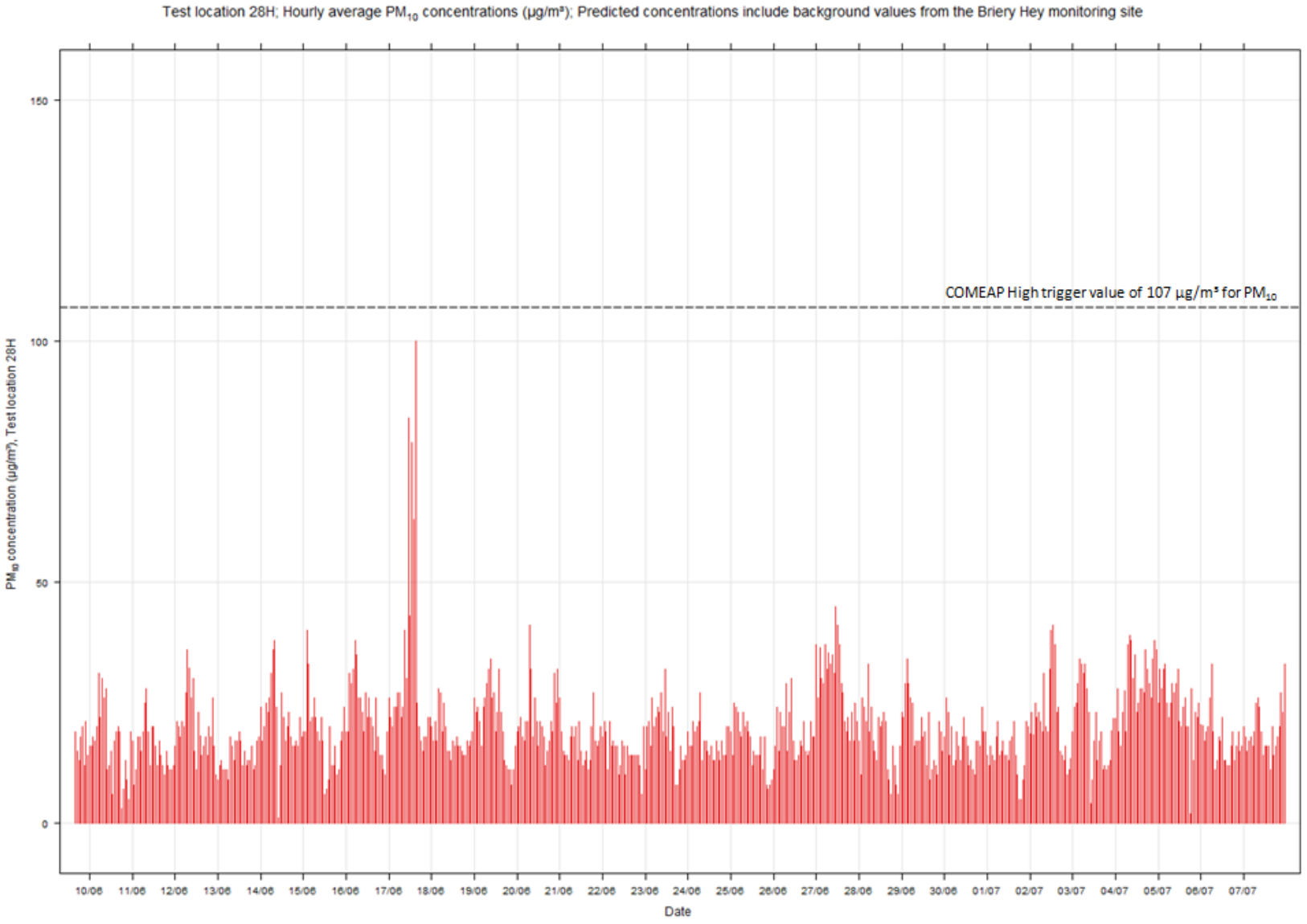

Test location 20; Hourly average PM10 concentrations (µg/m³); Predicted concentrations include background values from the Briery Hey monitoring site
COMEAP High trigger value of 107 µg/m³ for PM10
PM10 concentration (µg/m³), Test location 20
150
100
50
0
10/06
11/06
12/06
13/06
14/06
15/06
16/06
17/06
18/06
19/06
20/06
21/06
22/06
23/06
24/06
25/06
26/06
27/06
28/06
29/06
30/06
01/07
02/07
03/07
04/07
05/07
06/07
07/07
Date


Test location 23; Hourly average PM10 concentrations (µg/m³); Predicted concentrations include background values from the Briery Hey monitoring site
COMEAP High trigger value of 107 µg/m³ for PM10
PM10 concentration (µg/m³), Test location 23
150
100
50
0
10/06
11/06
12/06
13/06
14/06
15/06
16/06
17/06
18/06
19/06
20/06
21/06
22/06
23/06
24/06
25/06
26/06
27/06
28/06
29/06
30/06
01/07
02/07
03/07
04/07
05/07
06/07
07/07
Date


Test location 24; Hourly average PM10 concentrations (µg/m³); Predicted concentrations include background values from the Briery Hey monitoring site
COMEAP High trigger value of 107 µg/m³ for PM10
PM10 concentration (µg/m³), Test location 24
150
100
50
0
10/06
11/06
12/06
13/06
14/06
15/06
16/06
17/06
18/06
19/06
20/06
21/06
22/06
23/06
24/06
25/06
26/06
27/06
28/06
29/06
30/06
01/07
02/07
03/07
04/07
05/07
06/07
07/07
Date


Test location 25; Hourly average PM10 concentrations (µg/m³); Predicted concentrations include background values from the Briery Hey monitoring site
COMEAP High trigger value of 107 µg/m³ for PM10
PM10 concentration (µg/m³), Test location 25
150
100
50
0
10/06
11/06
12/06
13/06
14/06
15/06
16/06
17/06
18/06
19/06
20/06
21/06
22/06
23/06
24/06
25/06
26/06
27/06
28/06
29/06
30/06
01/07
02/07
03/07
04/07
05/07
06/07
07/07
Date


Test location 27; Hourly average PM10 concentrations (µg/m³); Predicted concentrations include background values from the Briery Hey monitoring site
COMEAP High trigger value of 107 µg/m³ for PM10
PM10 concentration (µg/m³), Test location 27
150
100
50
0
10/06
11/06
12/06
13/06
14/06
15/06
16/06
17/06
18/06
19/06
20/06
21/06
22/06
23/06
24/06
25/06
26/06
27/06
28/06
29/06
30/06
01/07
02/07
03/07
04/07
05/07
06/07
07/07
Date


Test location 28H; Hourly average PM10 concentrations (µg/m³); Predicted concentrations include background values from the Briery Hey monitoring site
COMEAP High trigger value of 107 µg/m³ for PM10
PM10 concentration (µg/m³), Test location 28H
150
100
50
0
10/06
11/06
12/06
13/06
14/06
15/06
16/06
17/06
18/06
19/06
20/06
21/06
22/06
23/06
24/06
25/06
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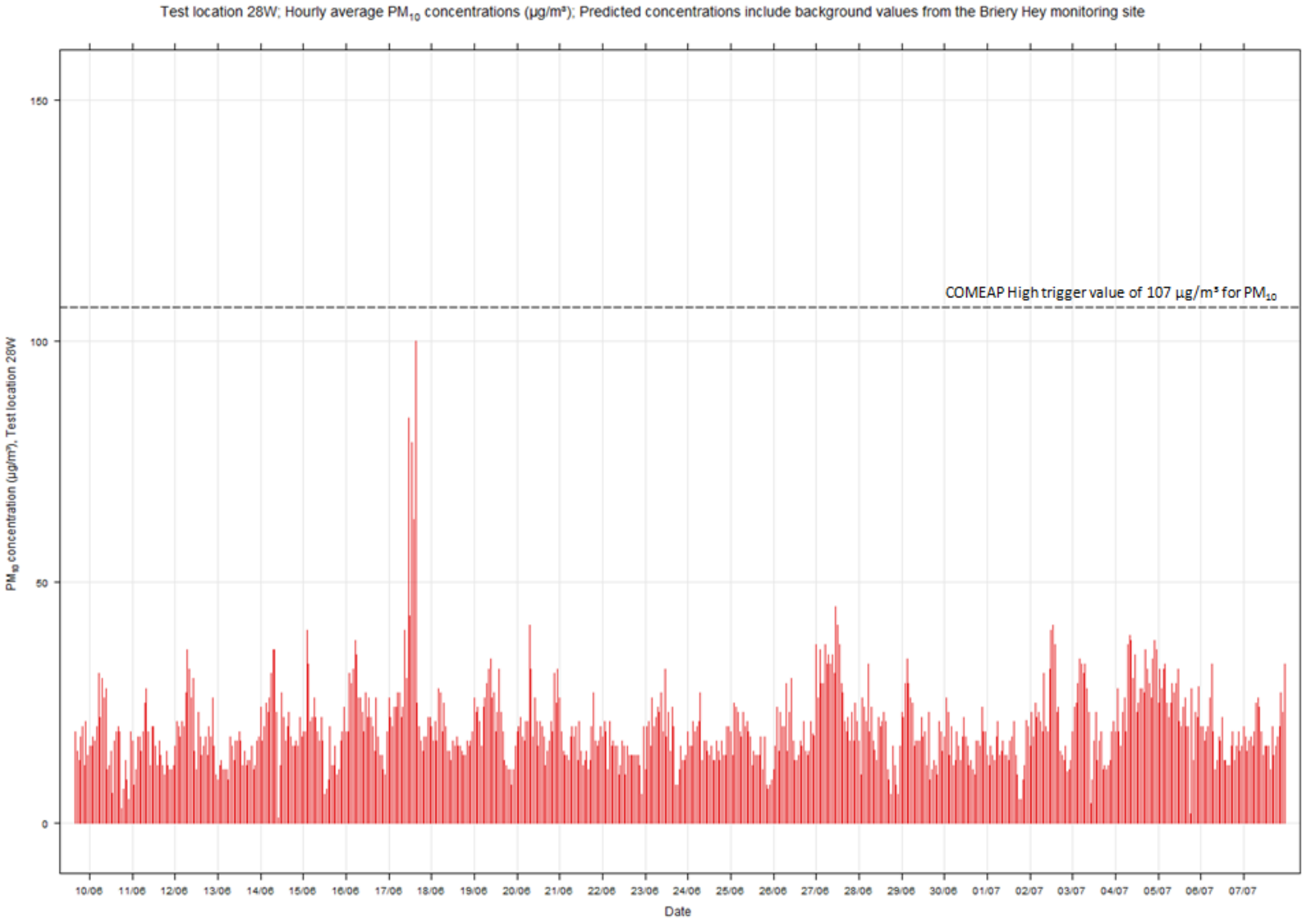
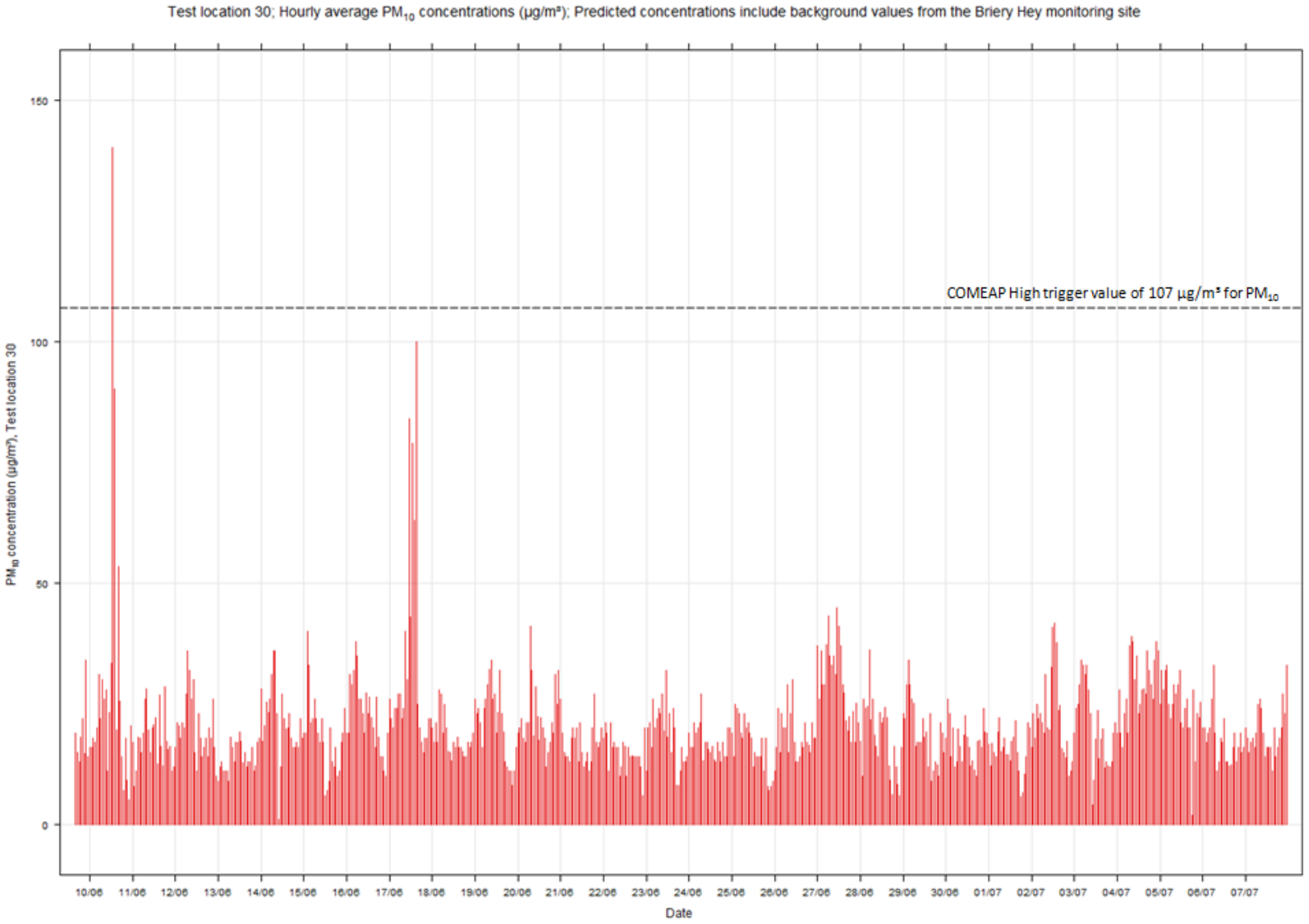
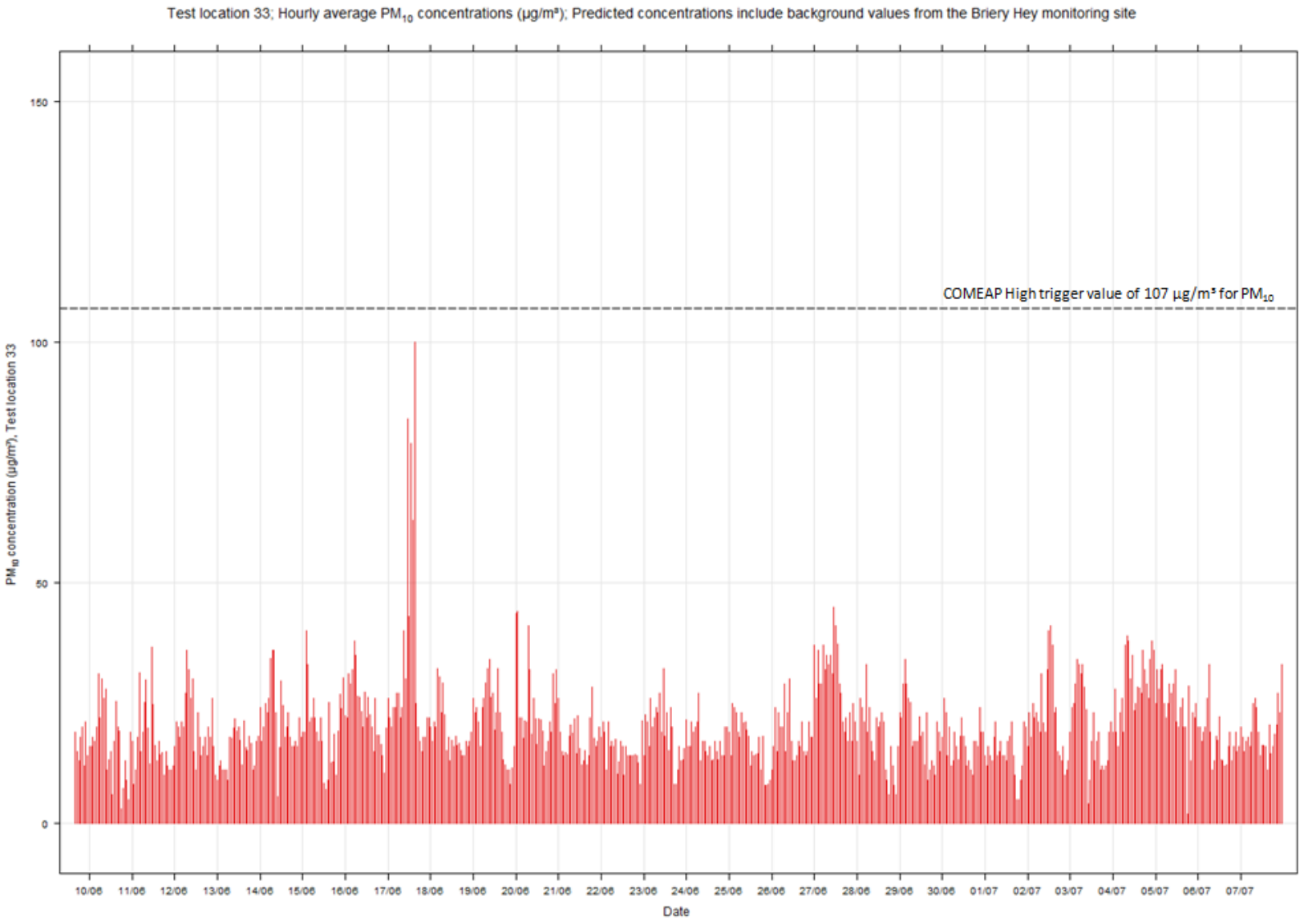
06/07
07/07
Date


Test location 28W; Hourly average PM10 concentrations (µg/m³); Predicted concentrations include background values from the Briery Hey monitoring site
COMEAP High trigger value of 107 µg/m³ for PM10
PM10 concentration (µg/m³), Test location 28W
150
100
50
0
10/06
11/06
12/06
13/06
14/06
15/06
16/06
17/06
18/06
19/06
20/06
21/06
22/06
23/06
24/06
25/06
26/06
27/06
28/06
29/06
30/06
01/07
02/07
03/07
04/07
05/07
06/07
07/07
Date


Test location 30; Hourly average PM10 concentrations (µg/m³); Predicted concentrations include background values from the Briery Hey monitoring site
COMEAP High trigger value of 107 µg/m³ for PM10
PM10 concentration (µg/m³), Test location 30
150
100
50
0
10/06
11/06
12/06
13/06
14/06
15/06
16/06
17/06
18/06
19/06
20/06
21/06
22/06
23/06
24/06
25/06
26/06
27/06
28/06
29/06
30/06
01/07
02/07
03/07
04/07
05/07
06/07
07/07
Date


Test location 33; Hourly average PM10 concentrations (µg/m³); Predicted concentrations include background values from the Briery Hey monitoring site
COMEAP High trigger value of 107 µg/m³ for PM10
PM10 concentration (µg/m³), Test location 33
150
100
50
0
10/06
11/06
12/06
13/06
14/06
15/06
16/06
17/06
18/06
19/06
20/06
21/06
22/06
23/06
24/06
25/06
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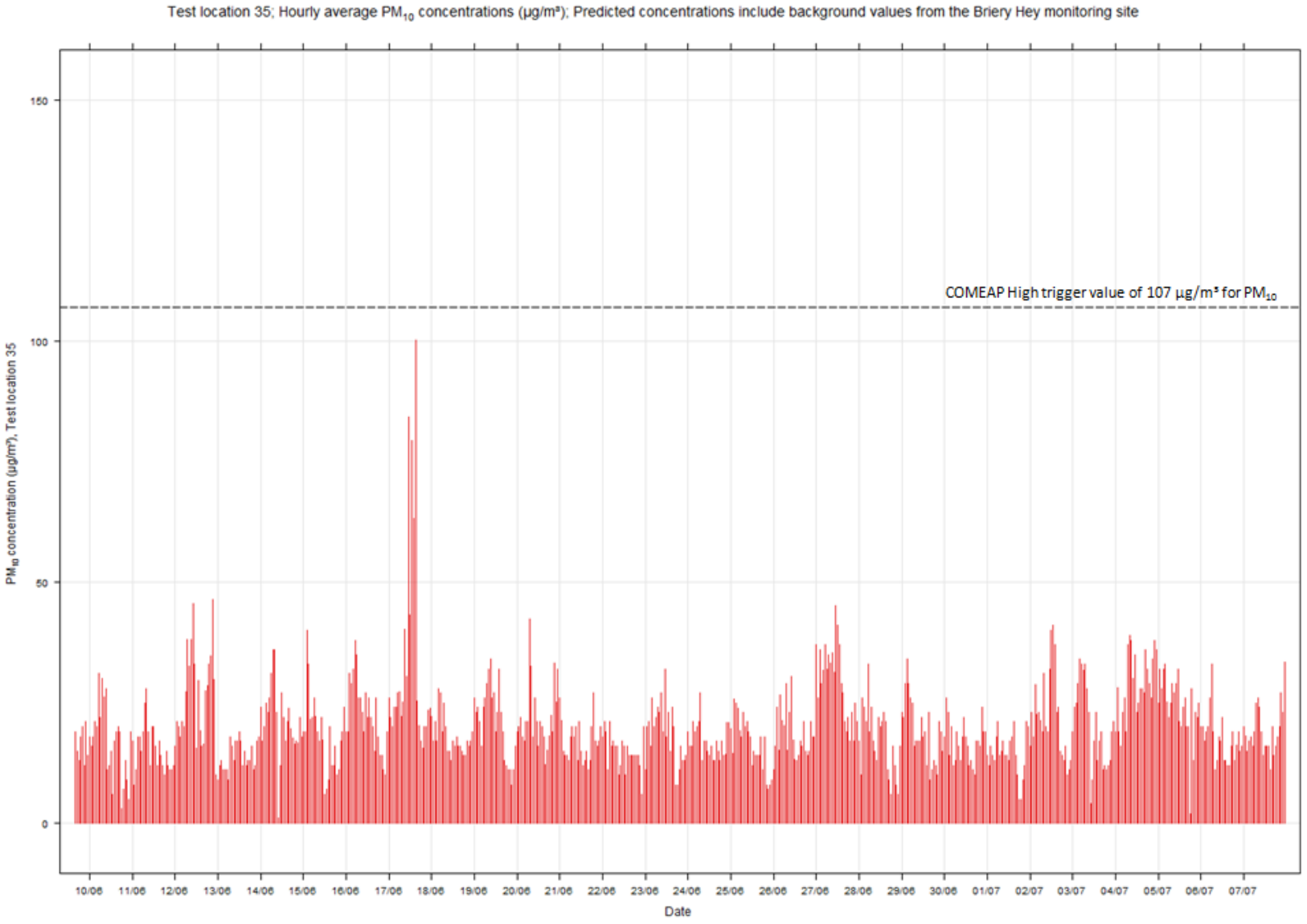
04/07
05/07
06/07
07/07
Date


Test location 35; Hourly average PM10 concentrations (µg/m³); Predicted concentrations include background values from the Briery Hey monitoring site
COMEAP High trigger value of 107 µg/m³ for PM10
PM10 concentration (µg/m³), Test location 35
150
100
50
0
10/06
11/06
12/06
13/06
14/06
15/06
16/06
17/06
18/06
19/06
20/06
21/06
22/06
23/06
24/06
25/06
26/06
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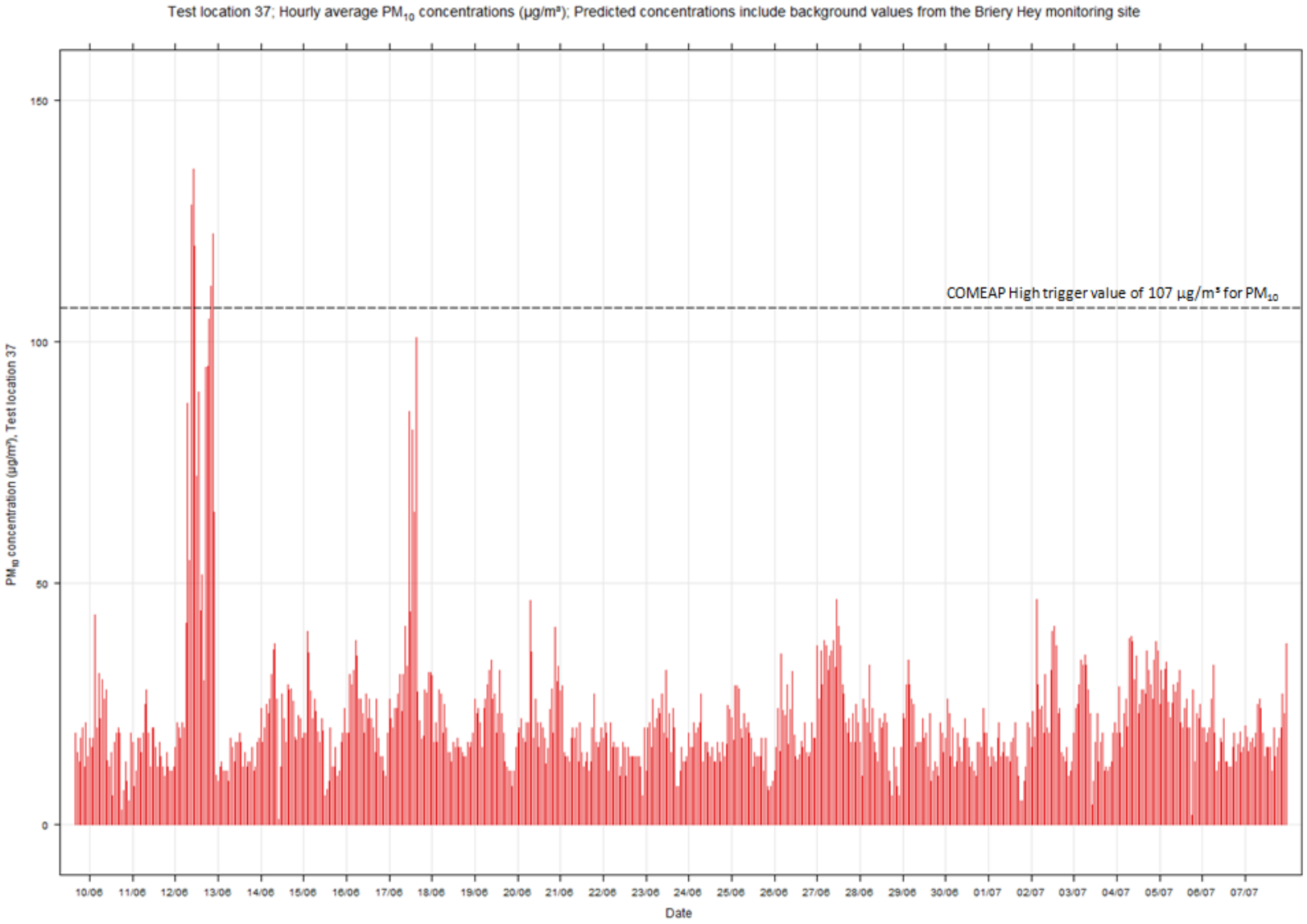
04/07
05/07
06/07
07/07
Date


Test location 37; Hourly average PM10 concentrations (µg/m³); Predicted concentrations include background values from the Briery Hey monitoring site
COMEAP High trigger value of 107 µg/m³ for PM10
PM10 concentration (µg/m³), Test location 37
150
100
50
0
10/06
11/06
12/06
13/06
14/06
15/06
16/06
17/06
18/06
19/06
20/06
21/06
22/06
23/06
24/06
25/06
26/06
27/06
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02/07
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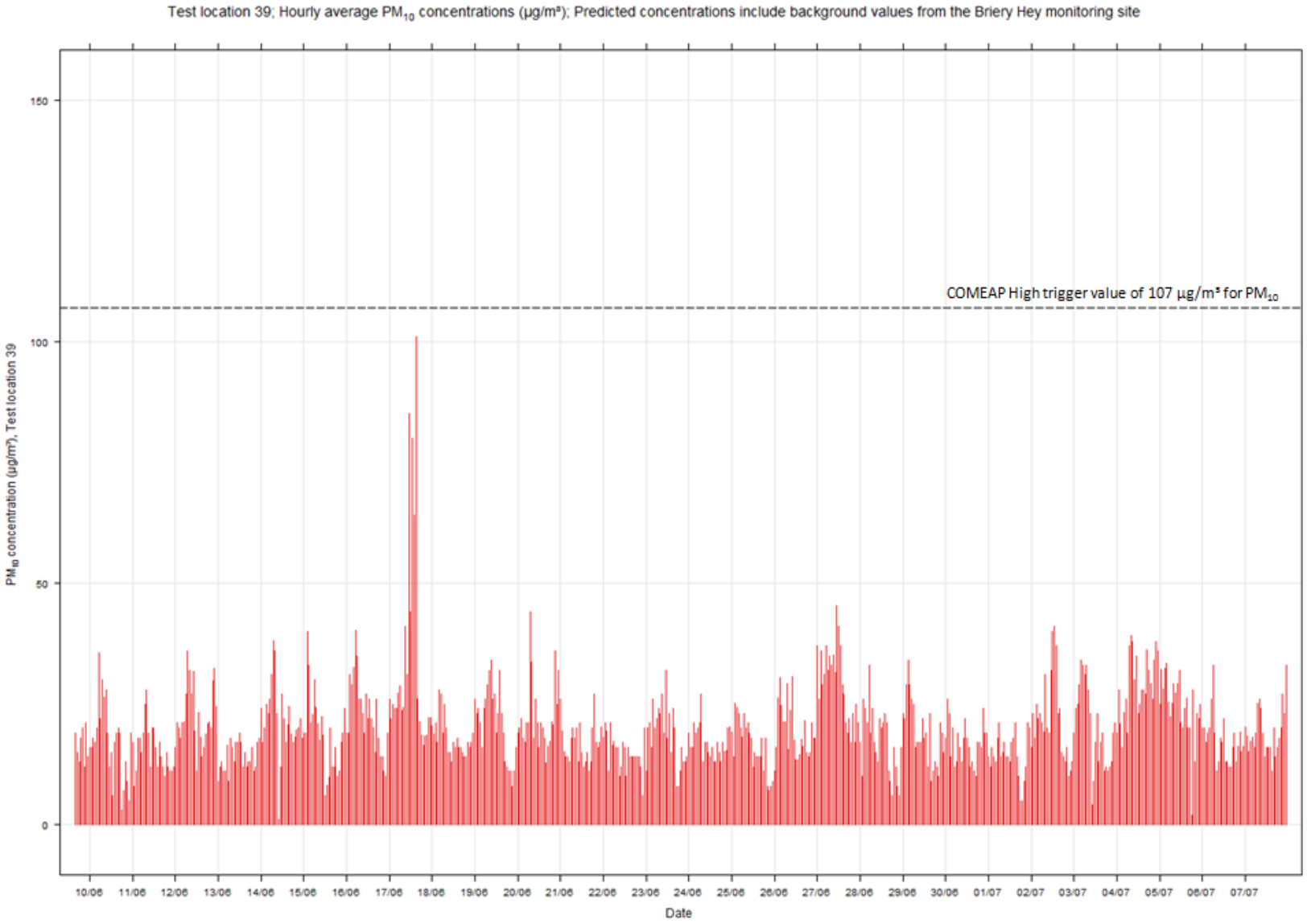
04/07
05/07
06/07
07/07
Date


Test location 39; Hourly average PM10 concentrations (µg/m³); Predicted concentrations include background values from the Briery Hey monitoring site
COMEAP High trigger value of 107 µg/m³ for PM10
PM10 concentration (µg/m³), Test location 39
0
50
100
150
10/06
11/06
12/06
13/06
14/06
15/06
16/06
17/06
18/06
19/06
20/06
21/06
22/06
23/06
24/06
25/06
26/06
27/06
28/06
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30/06
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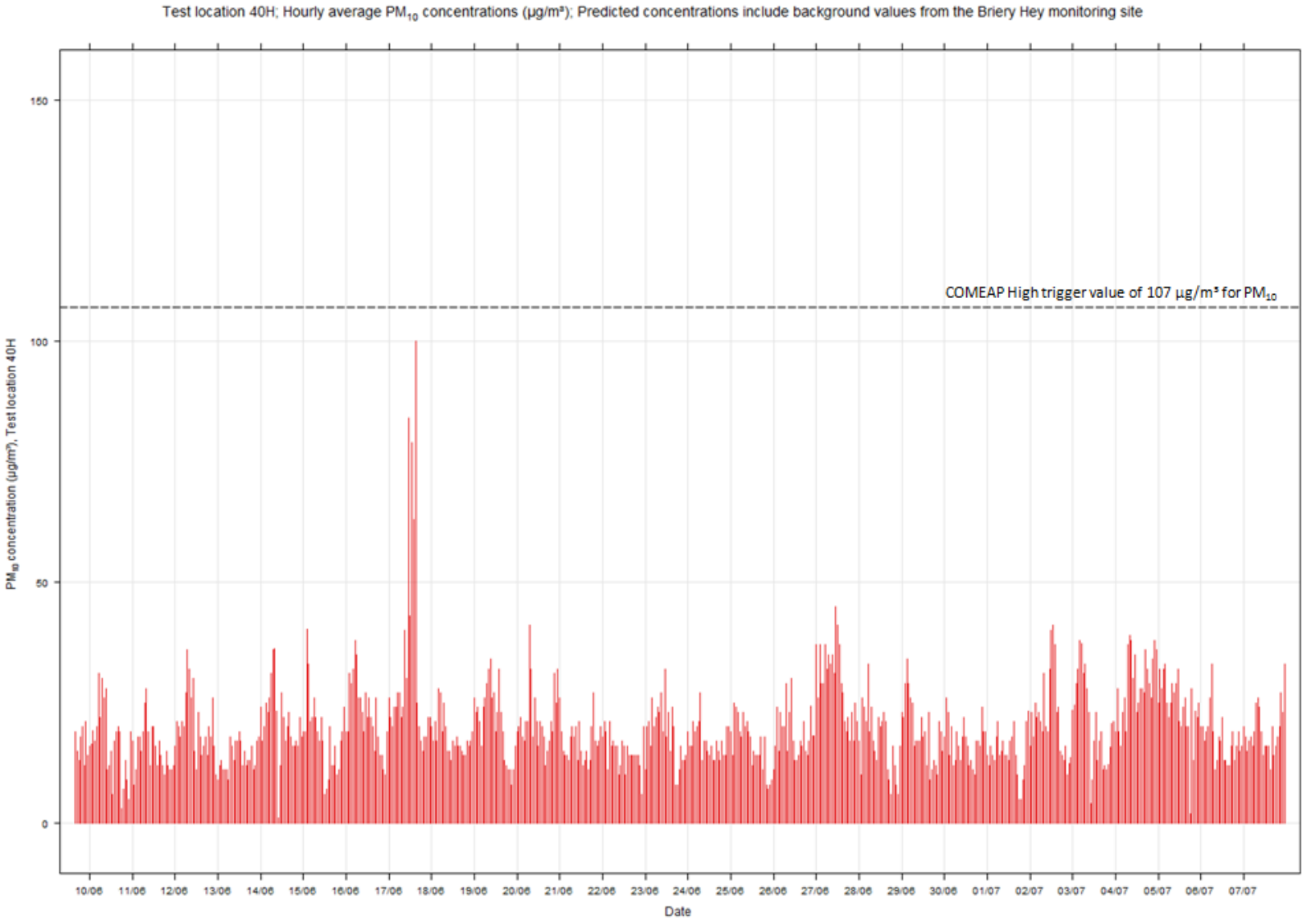
05/07
06/07
07/07
Date


Test location 40H; Hourly average PM10 concentrations (µg/m³); Predicted concentrations include background values from the Briery Hey monitoring site
COMEAP High trigger value of 107 µg/m³ for PM10
PM10 concentration (µg/m³), Test location 40H
150
100
50
0
10/06
11/06
12/06
13/06
14/06
15/06
16/06
17/06
18/06
19/06
20/06
21/06
22/06
23/06
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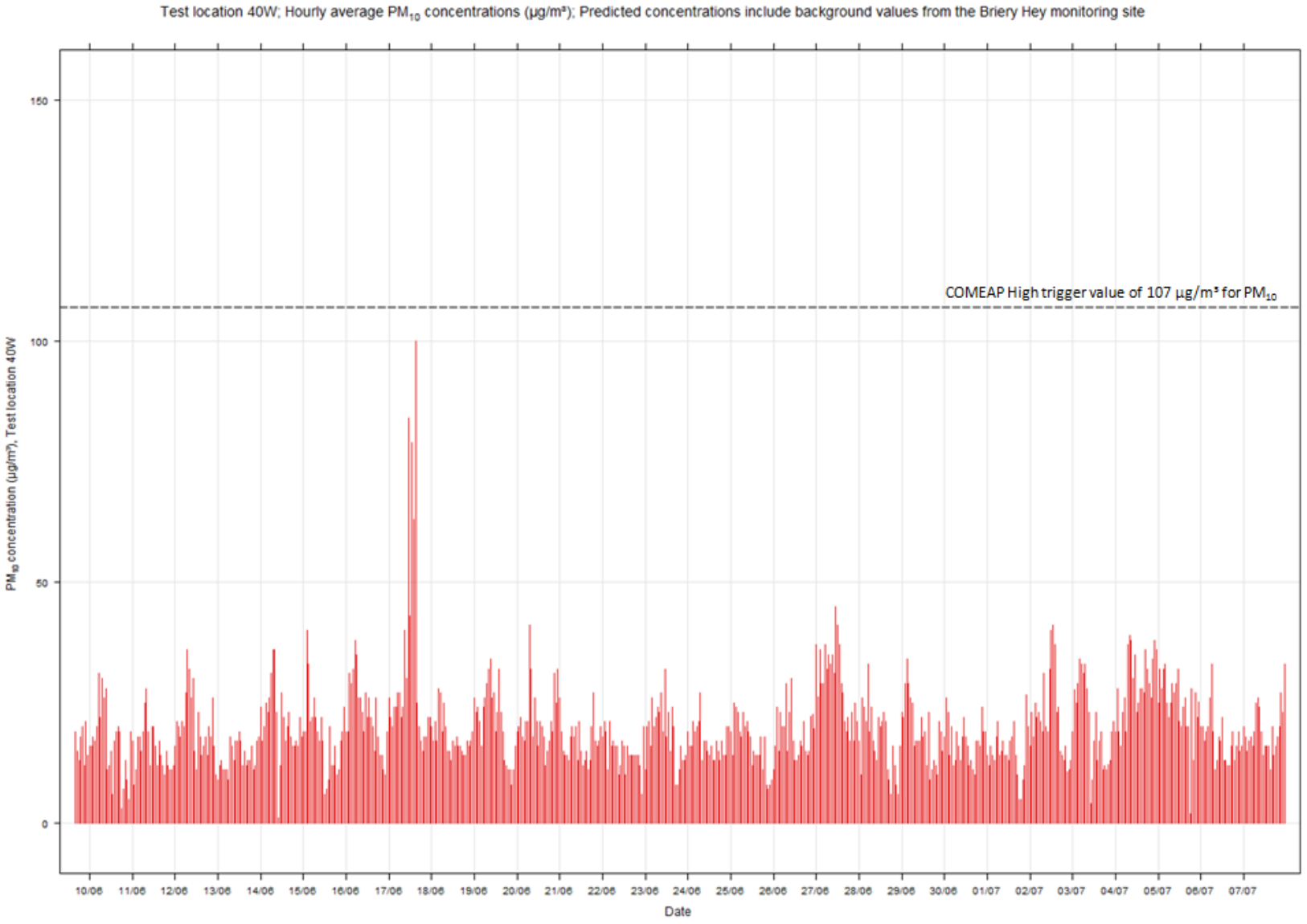
06/07
07/07
Date


Test location 40W; Hourly average PM10 concentrations (µg/m³); Predicted concentrations include background values from the Briery Hey monitoring site
COMEAP High trigger value of 107 µg/m³ for PM10
PM10 concentration (µg/m³), Test location 40W
150
100
50
0
10/06
11/06
12/06
13/06
14/06
15/06
16/06
17/06
18/06
19/06
20/06
21/06
22/06
23/06
24/06
25/06
26/06
27/06
28/06
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07/07
Date