

Koufos & Koufos v NTA [2013] NTSC 25

PARTIES: KOUFOS, Emmanuel (Manoli) &
BHNF KOUFOS, Attoumissas

v

NORTHERN TERRITORY OF
AUSTRALIA

TITLE OF COURT: SUPREME COURT OF THE
NORTHERN TERRITORY

JURISDICTION: SUPREME COURT OF THE
TERRITORY EXERCISING
TERRITORY JURISDICTION

FILE NO: 20815569

DELIVERED: 31 May 2013

HEARING DATES: 4 – 8 June, 12 – 15 June, 24 July, 12 –
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JUDGMENT OF: BLOKLAND J

CATCHWORDS:

NEGLIGENCE

Duty to take reasonable care – Duty owed to prospective tenants to provide habitable premises – Whether ongoing breach of duty occurred as to fitness and habitability of property – Reasonable regime of inspection established by defendant – Reasonable maintenance practices shown – no breach shown – *Residential Tenancy Act* (NT)

NEGLIGENCE:

Duty to take reasonable care – Defendant put on notice as to possible risk – whether response to notice reasonable – shortcoming in timeliness of response – defendant failed to act reasonably in response to notice as to risk – failure constitutes breach – breach unconnected to harm suffered by plaintiff

NEGLIGENCE

Causation – Foreseeability of risk – Whether source of contamination attributable to harm suffered – whether exposure to contamination attributable to harm suffered – plaintiff’s symptoms pre-existed the alleged causal nexus – plaintiff’s case fails on the issue of causation.

Evidence Act (NT) s 26

Residential Tenancies Act (NT) Part 5 – Division 1, s 25, Part 7 – Division 1, s 57, s 58,

Jones v Bartlett (2000) 205 CLR 166; *Watts v Rake* (1960) 108 CLR 158; *Wyong Shire Council v Shirt* (1980) 146 CLR 40; referred

Cavalier v Pope [1906] AC 428; *Donoghue v Stevenson* [1932] AC 562; *Northern Sandblasting Pty Ltd v Harris* (1997) 188 CLR 313; *Mackenzie v R* [1996] HCA 35; *Parker v South Australian Housing Trust* (1985) 41 SASR 493; *Roads and Traffic Authority of NSW v Dederer* (2007) 234 CLR 330; *Seltsam Pty Ltd v McGuinness and Anor* [2009] 49 NSWLR 262; cited

REPRESENTATION:

Counsel:

Plaintiff:	Mr Cranitch SC, Mr Campbell/Mr Malouf
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Defendant:	Mr Livesey QC, Ms Brownhill
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Solicitors:

Plaintiff:	Lloyd Lancaster
Defendant:	De Silva Hebron Barristers and Solicitors

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IN THE SUPREME COURT
OF THE NORTHERN TERRITORY
OF AUSTRALIA
AT DARWIN

Koufos and Koufos v NTA [2013] NTSC 25
No. 20815569

BETWEEN:

**Emmanuel (Manoli) Koufos &
BHNF Attoumissas Koufos**
Plaintiff

AND:

The Northern Territory of Australia
Defendant

CORAM: BLOKLAND J

JUDGMENT

(Delivered 31 May 2013)

Introduction

- [1] This action is brought by Mrs Attoumissas Koufos as the next friend of the plaintiff, her infant son Emmanuel (Manoli)¹ Koufos. The plaintiff alleges the Northern Territory² was negligent as the landlord of his family's residence at 35 Witherden Street Nakara (the premises).
- [2] The trial concerned the question of liability only.³

¹ Many witnesses referred to the plaintiff by the shortened form of his name 'Manoli'. Throughout these reasons the plaintiff is referred to variously as the plaintiff, "Emmanuel" or "Manoli".

² At the material time 'Territory Housing' was the division of the Department responsible for the provision of public housing in the Northern Territory. Throughout these reasons the defendant is referred to as both the "Northern Territory" and "Territory Housing".

³ Order of Master Luppino, 5 January 2012.

- [3] It is alleged that as a result of a breach of duty to ensure the premises were fit and habitable and not a danger to the tenants, Manoli suffered exposure to lead contamination on an ongoing basis and/or ingested lead from the soil of the premises. The claim in negligence is brought against the Northern Territory on the basis of the alleged breach of duty and asserted ongoing failures since 2005. It is claimed the negligence or breach of duty has caused both multiple medical conditions and permanent injury to Manoli. The plaintiff's case is that the injuries and conditions suffered by him are consistent with and caused by lead exposure.
- [4] The claimed injuries are lead poisoning, anaemia, behavioural problems, learning difficulties and developmental delay.⁴ Numerous other disabilities and conditions were also claimed to be caused by exposure to lead.⁵ The plaintiff's case in general is that as a result of the exposure to lead, Manoli developed symptoms consistent with or similar to autism, or within the spectrum of autism.
- [5] The defendant denies that it was a negligent landlord. The defendant acknowledges that one sample of the plaintiff's blood taken on 29 August 2006 revealed a lead level exceeding the reference range indicative of lead exposure for a child, but denies that the symptoms and medical conditions suffered by Manoli were caused by exposure to lead. If the plaintiff was

⁴ Amended Statement of Claim, para 16.

⁵ Amended Statement of Claim, para 17. Particulars of disabilities are delay in learning, nausea and vomiting, constipation and diarrhoea, hyperactivity, irritability, difficulties with speech, difficulties in communication generally, distractive behaviour, difficulties with organisational skills, difficulties with social skills, cognitive impairment, inability to interact and play with other children as would be expected of a person at Manoli's age.

exposed to lead as a result of negligence or any breach of duty on its part, the Northern Territory's case is that any such exposure did not cause injury to the plaintiff. It argues instead that to the extent the plaintiff suffered injuries or other conditions, temporary or permanent, they were suffered by him prior to taking up residence with his family at 35 Witherden Street; the injuries were not caused by his occupation at the premises. The defendant argues the evidence shows that Manoli's condition of autism and an intellectual disability pre-existed the move to the premises, although those conditions were not diagnosed at that time.

[6] The plaintiff's case is also put on the basis that after testing was carried out on the backyard soil of the premises on 31 October 2006 indicating abnormal limits of lead, Territory Housing failed to advise the plaintiff's parents of the results until 15 May 2007. It is claimed Manoli suffered ongoing exposure to lead between October 2006 and May 2007; that this was at a time that Territory Housing knew there was lead in the soil and that Manoli continued to be exposed by playing in the yard as his parents did not know of the lead levels in the soil. The case is put that the ongoing exposure to and ingestion of lead during this period was a breach of the defendant's duty of care and materially contributed to the plaintiff's injuries.

[7] The defendant argues the plaintiff's case, when put on this secondary basis, suffers from similar deficiencies, namely, (but not exclusively), the inability to prove any exposure resulted in the injuries or conditions suffered by the

plaintiff. The defendant argued there was no continued exposure to lead after soil samples were taken for testing on or about 31 October 2006.

General principles governing a landlord's duty of care

- [8] There is general agreement on the principles governing a landlord's duty of care. It is well accepted that landlords have a duty of care.⁶ *Northern Sandblasting Pty Ltd v Harris* confirmed earlier authority⁷ holding that immunity from liability in tort previously enjoyed by landlords⁸ is no longer the law in Australia. Liability must be determined according to the ordinary principles governing negligence.⁹
- [9] The duty of care requires a landlord to exercise reasonable care to avoid foreseeable risk of injury to tenants, members of the tenant's household and other persons who are lawfully on the premises.¹⁰
- [10] In *Jones v Bartlett*, the duty of care of a landlord was formulated by various members of the court in the following general terms: a duty owed to prospective tenants and members of their household to "take reasonable care to avoid foreseeable risk of injury";¹¹ "to take reasonable care to put and keep premises in a safe state of repair";¹² the landlord is obliged to take reasonable care to ensure that the premises do not suffer defects that the

⁶ *Parker v South Australian Housing Trust* (1985) 41 SASR 493; *Northern Sandblasting Pty Ltd v Harris* (1997) 188 CLR 313.

⁷ *Parker v South Australian Housing Trust* (above).

⁸ From the days of *Cavalier v Pope* [1906] AC 428.

⁹ *Parker v South Australian Housing Trust* (above) at 516-517 per King CJ; Prior J at 520 said "the power to sue in tort notwithstanding a contract is well established".

¹⁰ *Jones v Bartlett* (2000) 205 CLR 166 at [92], per Gaudron J.

¹¹ Gleeson CJ at [57] and [56]; citing Dawson J in *Northern Sandblasting v Harris* (cited above at 343).

¹² Gaudron J, at 155 [93].

landlord knows or ought to know make the premises unsafe.¹³ The premises must be fit for the purpose of their primary use, namely habitation as a domestic residence.¹⁴ As the occupier of premises is only required to take such care as is reasonable in the circumstances, “a landlord should not be subject to a higher duty to make premises as safe for residential uses as reasonable care and skill on the part of anyone can make them”.¹⁵

- [11] Although a landlord has a common law duty consistent with the general law of negligence, the content of the duty is affected by the lease arrangement and the fact that it is the occupier, rather than the landlord, who is in occupation of the premises.
- [12] Without specific terms provided in the lease, it is clear the content of the landlord’s duty, consistent with providing habitable premises for a domestic tenancy, includes a duty to inspect at the commencement of the tenancy.¹⁶ A landlord is not required, in the usual course, to appoint experts to inspect the premises and is required to remedy only those defects which may produce harm to people using the premises in an “ordinary way”.¹⁷ The content of the duty includes a duty to avoid harm caused by defects about which the

¹³ Gummow and Hayne JJ at 171-175, citing *Donoghue v Stevenson* [1932] AC 562; at 172, adopting Dawson J in *Northern Sandblasting v Harris*, a duty “to take reasonable care to avoid foreseeable risk of injury to the respondent”.

¹⁴ Gummow and Hayne JJ at [173].

¹⁵ Gaudron J at [92].

¹⁶ *Northern Sandblasting Pty v Harris* (1997) 188 CLR 313 at 587 per Brennan CJ; at 605-6 per Gaudron J.

¹⁷ *Jones v Bartlett* (2000) 205 CLR 166 at [178] per Gummow and Hayne JJ.

landlord knew, or should have known, when those defects arise during the course of the tenancy.¹⁸

[13] Both the Residential Tenancy Agreement between the parties¹⁹ and the *Residential Tenancies Act* operate to reflect and define the content of the duties of both parties in this case.

[14] Clause 8 of the Residential Tenancy Agreement relevantly provides:

8.1 The Landlord must ensure that the premises and ancillary property:

- (a) are habitable;
- (b) meet all health and safety requirements specified under an Act that apply to residential premises or the ancillary property; and
- (c) are reasonably clean when the tenant enters into occupation of the premises.

8.2 Subject to Part 7 of the RTA, the Landlord:

- (a) must ensure that the premises and ancillary property are in a reasonable state of repair when the Tenant enters into occupation of the premises; and
- (b) must maintain the premises and ancillary property in a reasonable state of repair, having regard to their age, character and prospective life.

8.4 The Landlord may in accordance with Clause 15 have its servants or agents enter the premises to effect any repairs and

¹⁸ *Northern Sandblasting Pty v Harris* (cited above) at 313 per Brennan CJ; *Jones v Bartlett* (cited above) at [88] per Gaudron J; at [173] – [179] per Gummow and Hayne JJ.

¹⁹ Exhibit 36, Agreed Tender Book 209-227.

maintenance and, if such repairs or maintenance result from the Tenant's failure to maintain the premises, as provided in Clause 8.5 hereof, the Landlord shall be entitled to recover from the Tenant all costs incurred by it by reason of such repairs or maintenance.

[15] Under the *Residential Tenancy Act* (NT) Part 5 – Division 1, a “Property Condition Report” is required at the commencement of the tenancy.

[16] Unless not practical, the landlord is to fill out the Property Condition Report in the presence of the tenant.²⁰ It might therefore be assumed that an inspection will take place in the ordinary course. Where the provisions of Part 5, Division 1 have been complied with, (that is, where the landlord gives a tenant a signed condition report, and the tenant has either accepted it or modified it in accordance with the provisions), the report is “conclusive evidence” of the condition of the premises at the beginning of the tenancy. Whether Mrs Koufos modified the relevant Property Condition Report drawing attention to the presence of a lead contaminant or possible lead contaminant and gave it to Territory Housing is a contentious matter and is discussed later in the reasons.²¹

[17] If through an inspection, a landlord, (here, the Northern Territory), knows or ought to know of any dangerous defects, the landlord is required to remedy them. The duty with regard to dangerous defects is discharged if the landlord takes reasonable steps to ascertain the existence of any such defects and, once the landlord knows of any, takes reasonable steps to remove them

²⁰ Section 25(3) *Residential Tenancy Act*.

²¹ Paras [31] – [40].

or to make the premises safe. This does not amount to a proposition that the ordinary use of the premises for the purpose for which they are let must not cause injury; it is that the landlord must act in a reasonable manner to remove the risks. If a landlord fails to remedy dangerous defects, they may be liable on the ground of negligent failure to assess and remove dangerous defects.²²

[18] It is not until the tenant assumes exclusive possession of the premises that the tenant acquires the status of occupier and the landlord loses a certain degree of control over the premises. This accords with the general reasoning in *Jones v Bartlett* already discussed.

[19] The duty of the landlord is invoked when a tenant notifies a landlord of the need of any repair because of a defect discovered or arising during the course of the tenancy. The landlord is required to remedy the defect if notified by the tenant. Effectively these obligations are reflected in ss 57 – 58 *Residential Tenancy Act* (NT) which in turn operate as terms of the tenancy.²³

The general history of the plaintiff and his family prior to the move to the premises at 35 Witherden Street.

[20] Much of the general history before the Court about Manoli and his family was received through the evidence of his mother Attoumissas Koufos²⁴ (Mrs Koufos). For reasons explored throughout this judgement, I approach the

²² Essentially reflected in the provisions of the *Residential Tenancy Act* (NT) Part 7 – Division 1. Section 57(1) *Residential Tenancy Act* (NT).

²⁴ Mrs Koufos also uses her former surname Mailis. As the action is brought in the name of Koufos, respectfully “Koufos” is used throughout these reasons.

evidence of Mrs Koufos with caution. Undoubtedly Mrs Koufos deals with significant family commitments. She cares for Manoli who clearly suffers severe disabilities. She is an attentive mother. Some of her evidence was, however, lacking in credibility and reliability in fundamental ways. Those parts of her testimony diminished the standing of her evidence as a whole. Unless supported by other credible evidence I cannot rely in any significant way on the evidence of Mrs Koufos, especially on contentious points. When her evidence conflicts with other reliable and credible evidence, I prefer that evidence over the evidence of Mrs Koufos.

[21] Manoli was born on 4 July 2002. Mrs Koufos has two children with a previous husband, (Mr Koumborous), Michalas (born July 1996) and Aphrodite (born January 1998). She has two younger children with Mr Panormitis Koufos, Manoli's father. As well as Manoli, the children Mrs Koufos has with Mr Koufos are Panayotis (born October 2003) and Pantelys (born July 2005). She and Mr Koufos are no longer together, but when they were, two children from his previous relationship regularly stayed with them. Prior to, and on moving into the premises at 35 Witherden Street, there were often five children in the home. Pantelys was born shortly after the family moved to the premises at Witherden Street.

[22] The evidence also shows that at various times Mrs Koufos was under

significant stress.²⁵ At times there was conflict of some significance between Mrs Koufos and her husband, (although Mrs Koufos said the family dynamics were normal, no conflicts²⁶), including reported violence directed towards her;²⁷ and at times Mr Koufos was away from the family due to work.²⁸

[23] The family's general circumstances, and Manoli's in particular, are important factors bearing on the issue of whether Manoli began to experience symptoms, or was observed with symptoms consistent with either lead exposure or indicative of autism, intellectual disability or any of these prior to the family moving into the premises in June 2005; or whether any of these conditions or symptoms manifested only after the move.

[24] A significant element underpinning the plaintiff's case is the assertion that apart from some minor unrelated health issues that resolved with minimal treatment, Manoli and the rest of the family were healthy until soon after they moved into the premises; Manoli's health was said to deteriorate after then. This is said to lend weight to the plaintiff's case that exposure to and/or ingestion of lead at the premises caused the conditions and the disabilities that regrettably Manoli suffers from. A further consideration is whether Manoli possessed a particular vulnerability to lead, and if he was

²⁵ Eg. 2002, T 69-70.1. When Pantelys was eight or nine months old, Mrs Koufos confirmed tiredness, being run down, loss of hair during breast feeding. Confirmed in the notes of the Community Care Clinic. Dr Duff reported a history of familiar stress. (T 432). Dr Kilham was concerned maternal stress was part of the behaviours. (T 635). See also Dr Marraffa at 809. T 22.

²⁶ 2005, 2006, T 230-232, 290, 291.

²⁷ 2005, 2006, T 230-232, 290, 291.

²⁸ Mrs Koufos said this occurred only once, for three or four days; Mr Koufos said he was away for one month, in 2007; R 219, 281.

subject to continual exposure, whether such exposure caused or materially contributed to his disabilities.

[25] Prior to moving into 35 Witherden Street, Mrs Koufos said the health of the family was very good. She told the court her daughter Aphrodite had a speech delay and had received support from a teacher. The court was told Aphrodite was 14 at the time of the trial, she had some difficulty with writing, however Mrs Koufos said she no longer had speech problems or relationship issues. Mrs Koufos told Dr Marraffa, the consultant paediatrician called by the defendant, (although initially consulted by the plaintiff's former solicitor), that Aphrodite had no language at seven years of age; that Panayotis had problems running and breaking things, and had poorly constructed behaviour and problems in transition at school.²⁹

[26] Mrs Koufos said her brothers, Charlie and Michael also had speech delay. She denied her sister's eldest children had speech problems and denied telling Dr Marraffa the same. Mrs Koufos' brother, Charlie Mailis told the court that Maria's³⁰ son Dimitri had speech problems. Michael Mailis told the court his sister Maria's daughter and her son Dimitri had speech problems.³¹ This general history has some significance given the plaintiff argues Manoli's speech delay would have resolved as was the case for the rest of the family members who have, or had, this problem at earlier times in their lives. The evidence given by Mrs Koufos on this subject is but one

²⁹ T 813.9.

³⁰ Mrs Koufos' sister.

³¹ T 212-213, 374, 398.

example of inconsistencies between her evidence and the evidence of others called in the plaintiff's case. In her testimony she disagreed with the various histories recorded by doctors, producing further overall inconsistencies. For Dr Marraffa and Dr Kilham, (whose opinions about Manoli's condition are discussed below), delayed speech was a significant indication of developmental delay leading in part to a diagnosis of autism. Their opinions excluded lead as contributing to Manoli's condition. Manoli's physical health and observed behavioural and developmental history are discussed later in these reasons.³²

Initial approach to Territory Housing by Mrs Koufos and the offer of the tenancy of the premises

- [27] Prior to moving into the premises, the Koufos family were given notice to vacate their previous privately rented premises. When Mrs Koufos contacted Territory Housing at the end of May 2005 and explained the family circumstances, (including that she was pregnant and cared for a number of other children), she was given a priority application form.³³ Her first application for priority was not accepted at the end of May 2005. She made a complaint to the Ombudsman about this. She understood there was a 12 month waiting list at that time, even for urgent cases, but she did not think enough was being done for her at that time.³⁴

³² Paras [126] – [166].

³³ T 152, 22, 23.

³⁴ T 81-82.

[28] Mrs Koufos said she had been contacted by someone named “Jan”, from Territory Housing, (most likely, I accept was Ms Jean Saunders³⁵) in May 2005 about the Witherden Street premises becoming available. Mrs Koufos said she was told the premises would not be opened for her to see at that time, but she could drive by the premises and call back to indicate if she wanted the house.³⁶ Ms Saunders’ evidence was that it was normal practice to open a house for inspection, however, prospective tenants were also invited to “drive by” and say if they wanted the house from the “drive by”. In any event Mrs Koufos agreed there was nothing to prevent her from walking around the yard of the premises, she was not in a hurry.³⁷ In evidence in chief Mrs Koufos said she and her husband drove past the house, and said it was fine.³⁸ She said that although she did not go into the house, they stopped on the outside of the road, on the street driveway, parked, looked and then left the premises.³⁹ Mr Koufos said they did not have a look at the place. They drove past; they did not go into the premises.⁴⁰ Either the same day or the following day Mrs Koufos told Territory Housing that she wanted the house.

[29] I find there was nothing preventing the plaintiff’s parents from inspecting the property, had they wished to, prior to taking up the tenancy. The probabilities are that the usual practice of Territory Housing offering an

³⁵ Ms Saunders worked for Territory Housing for 19 years; as an allocations officer from October/November 2004 – August 2005; October/November 2006 and April 2007; T 722.

³⁶ T 24.

³⁷ T 82.

³⁸ T 24-25.

³⁹ T 82.

⁴⁰ T 272.

inspection or the alternative of a “drive by” were complied with. Given the circumstances of the Koufos family, it is likely the plaintiff’s parents were content to take the premises after seeing it from the outside.

[30] On 22 June 2005, Mrs Koufos met with Ms Jean Saunders at Territory Housing. Ms Saunders, Mrs Koufos and her husband signed the lease. There was a discussion about general obligations. Mr and Mrs Koufos were given keys allowing them access to the property. Ms Saunders completed records relating to rent and bonds and modes of payment of rent.⁴¹

The Property Condition Report for the premises

[31] At the same meeting on 22 June 2005, Mrs Koufos was given a copy of the Property Condition Report. The Property Condition Report noted an inspection on 7 June 2005 which reviewed the rear lawns and gardens.⁴² Mrs Koufos agreed Ms Saunders gave her the Property Condition Report when she signed the lease; she said Ms Saunders did not tell her it was important; she did not recall being told to go around the house and see whether she agreed with the Property Condition Report, although she remembered she was told to fill it in and bring it back to Territory Housing. At first she said she did not recall, and then said she did recall Ms Saunders saying to bring it back within five business days.⁴³ She agreed she was told that if she did not bring it back she would be taken to have accepted the

⁴¹ T 723-724.

⁴² Pratap Singh, affidavit sworn 26 April 2012, Annexure 1, Exhibit 30, 393-401.

⁴³ T 85.

condition of the property as set out in the report. She agreed that both she, Mr Koufos and Ms Saunders signed the Property Condition Report.⁴⁴

[32] Mrs Koufos says she returned the completed Property Condition Report to Territory Housing which recorded her amendments: her notation of rubbish, burnt batteries and a fire pit in the backyard of the premises. No such amended Property Condition Report with notations from Mrs Koufos was located in Territory Housing records. Territory Housing denies an amended Property Condition Report was returned by Mrs Koufos.

[33] Ms Saunders gave evidence of Territory Housing's practice: when tenants sign a Property Condition Report, their attention is drawn to the term that stipulates they have five days to return it, otherwise it is taken to be as noted by Territory Housing.⁴⁵ Ms Jan Wray's⁴⁶ evidence about general procedure in relation to Property Condition Reports is consistent with Ms Saunders' evidence. I accept the evidence given by Territory Housing officers about their procedures. I also accept the Property Condition Report as completed by Territory Housing is the file copy exhibited in these proceedings;⁴⁷ and that Mrs Koufos also obtained a further copy of it from Territory Housing when she was asked to do so by her lawyer. Mrs Koufos agreed she did not request her solicitor to write to Territory Housing to complain about not being given the Property Condition Report that she filled

⁴⁴ T 85-86; Exhibit 30 at 449.

⁴⁵ T 655-656.

⁴⁶ Currently Housing Manager, Darwin. She has held various positions with Territory Housing 2004 – 2007.

⁴⁷ Exhibit 30, pp 443-447.

in; she did not ask the Ombudsman to obtain a copy of the Property Condition Report she says she had filled in.⁴⁸

[34] Mrs Koufos maintained throughout her evidence that she filled in the tenant's part of the Property Condition Report in green pen and handed it in to the Casuarina Office of Territory Housing, to Ms Annie Warner. I do not accept Mrs Koufos' evidence about this. The evidence she gave about numerous particulars of everything she said she could remember noting on the Property Condition Report was severely lacking in credibility. Mrs Koufos would have the court believe that she could remember, almost line by line every item she had made a comment on. I will not set out the considerable volume of notations that Mrs Koufos said she could recall from June 2005; but I cannot accept her evidence about this.⁴⁹

[35] There is no doubt Mrs Koufos has had serious family matters to contend with. Perhaps she was being just silly about this evidence, not appreciating its importance. Senior counsel appearing for the plaintiff has submitted Mrs Koufos is not a sophisticated person.⁵⁰ I am prepared to accept that is the case and readily make reasonable allowances, however Mrs Koufos could not possibly have remembered the details of the Property Condition Report she gave evidence about. Her preparedness to give this testimony diminishes her evidence significantly. Given Mrs Koufos has long held the belief that Territory Housing are responsible for Manoli's condition, this

⁴⁸ T 124-128.

⁴⁹ T 89-105, 108-118, 120-122, 129-130.

⁵⁰ T 953.

may have shaped her perspective about what has occurred. This may explain in some way her defiant tone when giving certain evidence seemingly full of conviction but so obviously wrong. Mrs Koufos' evidence about having written "dirty and rubbish" and "burn marks" or various combinations of those words on the Property Condition Report is so confusing, internally inconsistent and unreliable that I reject what she has said about it. It also diminishes the credibility of her claim to have actually made such observations of the backyard.⁵¹

[36] Mr Koufos told the court he recalled his wife holding a piece of paper making a list, going around the house using a pen;⁵² she told him she had to go back to Territory Housing and ask for another form; that all he knew about that form was what she had told him.⁵³ This evidence, influenced as it is by what Mrs Koufos told him does not possess the quality of independence necessary to support Mrs Koufos' testimony. Mrs Koufos drafted Mr Koufos' statement. This diminishes the value of his evidence.

[37] I readily prefer the evidence of the officers of Territory Housing about all issues relevant to the Property Condition Report. I accept Ms Warner's evidence that she was aware of Mrs Koufos' allegation, (that she handed the Property Condition Report to her), and that she does not believe that allegation to be correct. She told the court she puts notes on the Tenancy Management System (TMS) when she has dealings with a tenant; there was

⁵¹ T 221, 113, 114-118.

⁵² T 274.

⁵³ T 284.

no reason why she would omit making a note; that she recalled speaking to Mrs Koufos about other matters to do with the tenancy; that she would record attendances and if she took a call from Mrs Koufos, would forward it on to the tenancy team. In cross-examination she agreed that if the management team of a particular tenancy was not available she would put the Property Condition Report in her tray. She agreed that doing so would not require an entry in TMS, however, in re-examination she said she would put notes on the system to say it had been received but she would not necessarily put notes on to say she had placed it in someone's tray; she would leave it at that and it would be obvious that it would be handed to the appropriate tenancy team.⁵⁴ It is clear Ms Warner accepted that errors were possible with respect to the operation of the TMS but she considered and understood the importance of a Property Condition Report. I accept her evidence that she would have noted its receipt and passed it on to the appropriate officer had it been given to her by Mrs Koufos.

- [38] Mr Pratap Singh, the relevant Tenancy Manager was not given a copy of a Property Condition Report with amendments by Mrs Koufos. Mr Singh told the court that if a form is brought to his attention and there are differences between the condition noted by the tenant and those of Territory Housing, an appointment is made to resolve the issue. Jan Wray told the court Mrs Koufos did not hand the property report to her. If she had done so, with notations that differed from the tenancy manager's report, she would have

⁵⁴ T 763.

made the appropriate arrangements. She emphasised the importance of this particular document and said if it raised an issue of maintenance of the premises, it would be wrong to just file it and not address the maintenance issue.⁵⁵

[39] I find it is highly unlikely that Mrs Koufos took a completed or amended a Property Condition Report and gave it to Territory Housing. Given the significance that Territory Housing placed on such reports, especially if it involved maintenance, it is highly unlikely that Territory Housing would have received it and misplaced it or did not in some way document its receipt. The more likely explanation is that Mrs Koufos did not amend and deposit an amended Property Condition Report because the condition of the property was reasonable without the need for contrary notations from her.

[40] As discussed earlier, under the relevant principles, a landlord must remedy a defect if notified by a tenant. The plaintiff cannot rely on an amended Property Condition Report as a notification to the landlord as there was no Property Condition Report with relevant notifications made.

The condition of the rear yard of the premises at the time the plaintiff moved into the premises

[41] The plaintiff's primary case relies on evidence alleging the presence of batteries, associated rubbish or ash, or other known contaminants in the yard at the time of the Koufos family moving in, or prior to that time. As batteries are known lead contaminants, the case is put that Territory Housing

⁵⁵ T 646-658.

not only knew of their presence but also knew of the resulting danger such a state of affairs was to the tenants.

[42] Before the court are photographs⁵⁶ depicting the state of the premises, including the lawns and gardens just prior to the Koufos family moving in. Mrs Koufos agreed they were a correct representation.⁵⁷ Just out of view of the photos of the yard, however, Mrs Koufos said there was a pile of rubbish; a burnt area; not a blazing fire, but ashes. It was 1.5 by 1.5 metres and was a circle.⁵⁸ She says she saw two batteries, a metal pole, some wooden chairs that were broken up and burned.⁵⁹ This area, or an area near to it was referred to at times in the proceedings as the “fire pit”, “sandpit” or “rubbish pit”. Mrs Koufos said the area was straight down from the clothesline, along the back fence; the fire pit was surrounded by bush rocks about 9 inches to a foot across;⁶⁰ she said there were little pebbles in the rubbish area.⁶¹

[43] Mrs Koufos said the rubbish included one tyre with a rim and rubber from car tyres. In relation to the two burnt batteries, one was complete in a box of normal size; the other was half a battery. Both were burnt. There were also plastic containers for chemicals and Kleenex chemicals. The wooden chair was broken into pieces and there were two dolls – they had a little hair on them; one a bit smaller than the other; there was also a metal pipe - she

⁵⁶ Exhibit D4.

⁵⁷ T 136.

⁵⁸ T 27.

⁵⁹ T 27.

⁶⁰ T 27.

⁶¹ T 155.

could not recall the length but it was 2 – 3 centimetres in diameter. There were other pieces of burnt plastic. They were about an inch and a half; Mrs Koufos said she could not say whether the number was closer to 12 or closer to 100. She did not see any tubing.

[44] Mr Koufos said he saw a plastic pipe and rocks in a circle formation. There were a mix of big and small rocks; some were a foot and some were half that size; there was plastic pipe in the circle. When asked if it was near the clothesline he initially said it was away from the clothes line.⁶² Mr Koufos said he started digging to see what was happening underneath. When he started to dig, he unearthed batteries, pipes and broken bottles. He said they were burnt. He was not sure if there were plastic dolls. He said it looked like the leg of a chair and a car tyre. He could not remember everything. He said there were two normal size car batteries; one was a full size battery, the other was burnt. It was while he was digging that he saw the bits of plastic and the batteries and that was when he saw the ash. He said it was “down low”. He said he called his wife to see what was in there. At that time a cousin, Ms Olga Fountis was going by and she came in.⁶³

[45] Mrs Koufos gave evidence that Mr Koufos was working on cleaning up the rubbish and the ash in the fire pit for approximately six to seven days.⁶⁴ Mr

⁶² T 273, 282.

⁶³ T 283.

⁶⁴ T 29, 155-156.

Koufos' evidence was that it took him about three hours to clean up the fire pit area which he did all on the one day.⁶⁵

[46] Although there are some similarities in their evidence as to some of the rubbish and batteries being in the back yard, there is a gross difference between seeing burnt batteries in a fire pit as Mrs Koufos suggested and digging down below and finding a number of articles including car batteries as suggested by Mr Koufos. There is a significant disparity in the evidence about the time taken to clean the firepit. Mr Koufos later in cross examination agreed the batteries were near the clothesline; or he couldn't remember.⁶⁶ Mr Koufos was clearly influenced by what Mrs Koufos told him.⁶⁷

[47] Ms Olga Fountis said she visited the family after they received the keys and saw Mr Koufos cleaning up because there was a lot of rubbish. She remembered paper, plastic, coke and fanta tins. She acknowledged she could not remember exactly, but generally she said she remembered there was rubbish. She said there were two pits. She said the father was cleaning the bigger pit that was surrounded by rocks.⁶⁸ Ms Fountis' evidence overall was not particularly reliable. Her evidence was not objective. At times it appeared she was prepared to give evidence about matters for which she either had no knowledge or had some knowledge but chose to give evidence in accordance with how she thought it might support the plaintiff's case.

⁶⁵ T 276, 283.

⁶⁶ T 290.

⁶⁷ T 289-290.

⁶⁸ T 359.

For example she denied the plaintiff had attended Royal Darwin Hospital prior to moving into the premises.⁶⁹ She denied the plaintiff's speech difficulties⁷⁰ and denied there were major arguments between Mr and Mrs Koufos.⁷¹ She also denied Mrs Koufos wanted money for Manoli.⁷² Ms Fountis spoke to Mrs Koufos about the court case when she signed her statement.⁷³

[48] I cannot accept the evidence about the state of the backyard and the visible presence of rocks, ash, batteries and other rubbish. Other credible evidence contradicts it. The previous tenant, Ms Sasha Moar confirmed the contents of the Property Condition Report that she filled out at the commencement of her tenancy. Ms Moar's evidence tends to confirm the evidence given about inspections and processes adopted by Territory Housing. In the course of her inspection of the premises, Ms Moar did not see any fire pits in the back yard. No burned areas, no batteries, no battery carcasses, pipes or other items of rubbish. She said if she had seen any of those things she would have made a note in the Property Condition Report because she did not want to pay for cleaning up someone else's mess.⁷⁴

[49] She acknowledged that when she inspected the property in March 2004 there was spear grass in the yard. The spear grass was tall and the yard was full of it. She disagreed that it was possible there may have been things that she

⁶⁹ T 358, 363.

⁷⁰ T 358, 364.

⁷¹ T 362.

⁷² T 367.

⁷³ T 368.

⁷⁴ T 773.

had overlooked. She said she walked over every inch of the property. She denied there may well have been a burnt patch that she had overlooked.⁷⁵ She said that while she was a tenant, two general inspections were carried out at the property. At neither of the inspections were there fire pits or plastic or burnt batteries or anything of that kind sticking out of the ground.⁷⁶ She walked with the property officer when the property inspections occurred. She said she had a positive recollection of walking around the entire property with Mr Pratap Singh. She said no fires were lit in the back yard when she was a tenant.

[50] Ms Moar agreed she did not go outside every day, however, she said apart from hanging clothes on the line she would go outside to see and feed her dog. She also put washing out on the clothing hoist every second day. Ms Moar said she was not happy to leave the premises as she was trying to buy it. It made her angry that she had to leave the property. She said that when she left the property in April 2005, its condition was similar to that shown in the photographs before the court.⁷⁷ She said she did not have to pay for any repairs or maintenance and was present for the final inspection with the tenancy manager Mr Pratap Singh. When she left the premises there was no fire pit in the back yard, no hole or burnt areas or battery carcasses or other items. She told the court Mr Singh met her at the house, she handed over

⁷⁵ T 784.

⁷⁶ T 774.

⁷⁷ Exhibit D4.

the keys and he did a walk through inspection. She said he told her he would let her know if there were any issues.⁷⁸

[51] Territory Housing's stated general policy and practice of cleaning up rubbish is manifest in previous examples of that type of work being undertaken. For example, in his statement, Pratap Singh refers to an additional work item relating to the premises in 2004 "remove bricks from front door and loose rubbish from yard". This work was completed at a cost of \$32.23.⁷⁹

[52] It is possible that batteries and/or other rubbish or contaminants were dug up by Mr Koufos from "down low", but I find that any objects meeting that description were not visible on the surface of the backyard of the premises at the time the plaintiff and his family moved into the premises. Those objects could not have been discovered by reasonable inspection. Neither were they seen during the many previous inspections of the property. Those inspections were well documented.⁸⁰

[53] The evidence of the previous occupant (Ms Moar) to the effect that there was no fire pit or batteries is convincing. Although I bear in mind Ms Moar was giving evidence of observations of some seven years before, there is other evidence, including property inspection reports, supportive of her recollection. While I accept that with some witnesses the firmness of the

⁷⁸ T 786.

⁷⁹ Affidavit, Pratap Singh, 26 April 2012, Exhibit D 30 at 318, para 34.

⁸⁰ Statement of Wally Jenkins, 16 March 2012; Affidavit of Pratap Singh, 26 April 2012, Exhibit D 30 at 318, para 34.

recollection belies the reliability of it,⁸¹ Ms Moar had no reason to give favourable evidence in respect of Territory Housing. On the contrary, she was not happy Territory Housing ended the tenancy.

[54] After Ms Moar's vacation of the premises, there were four further inspections undertaken by or on behalf of Territory Housing.⁸² Two of those inspections reviewed the rear lawns and garden. No report identifies a "fire pit" or evidence of batteries, rubbish or other identifiable contaminants.

[55] A Ms Edwards was to be a tenant of the premises on or about May 2005. Mr Pratap Singh conducted an inspection of the property on 10 May 2005 for the purpose of her tenancy. Ms Edwards did not ever go into occupation of the property. Before the Koufos family moved in, Mr Singh conducted another inspection on 7 June 2005 after contractors had completed work on the premises.⁸³ Mr Singh gave detailed evidence about a number of inspections. He told the court that if he had seen any fire pit or batteries, (which he did not), he would have given instructions to a contractor to have them removed.⁸⁴ These premises, including the yards, were in good condition. The probabilities are that Mr Singh would have noted and taken action on any visible contaminant or hazard.

⁸¹ Senior counsel for the plaintiff made this submission on the basis of *Mackenzie v R* [1996] HCA 35 "A person who is making a true mistake carrying that mistake through with his conviction he is correct."

⁸² 20 April 2005, Exhibit 30 at 371; 10 May 2005, Exhibit 30 at 373; 6 June 2005, Exhibit 30 at 391; 7 June 2005, Exhibit 30 at 393-401.

⁸³ T 679.

⁸⁴ T 677-695.

[56] In my view there was nothing at the time of moving into the premises, nor prior to the Koufos family taking up residence that could have reasonably put Territory Housing on notice of the presence of lead at a level that could injure tenants.

[57] Mrs Koufos did not disclose the presence of batteries in circumstances that such a disclosure might be expected. Although Dr Schempp⁸⁵ records Mrs Koufos telling her Emmanuel ate paint from the walls, Dr Schempp does not record any conversation about being told there were batteries where he played.⁸⁶ Mrs Koufos said that she did tell Dr Schempp about batteries.⁸⁷ Mrs Koufos also said she told Dr Schempp about batteries in November 2006 and February 2007, but this is not recorded as might be expected.⁸⁸ She said she mentioned batteries to Dr Edwards in March 2007.⁸⁹ Such a disclosure does not appear in the records.⁹⁰

[58] After she was referred to Dr Kilham's report of 11 July 2007,⁹¹ in which Dr Kilham raised the possibility himself of "an old battery on the site" as a possibility to explain high lead levels in the soil, Mrs Koufos answered questions in cross examination as follows:⁹²

I suggest that this was the very first time that – from this time you then started mentioning batteries to doctors? Yes.

⁸⁵ Paediatric Consultant, then at, Royal Darwin Hospital.

⁸⁶ Exhibit 36, Agreed Tender Book, 482-483, 705-707.

⁸⁷ T 159.

⁸⁸ T 162, 164; Exhibit 36, Agreed Tender Book, 703-704.

⁸⁹ T 164-165.

⁹⁰ Exhibit 36, Agreed Tender Book, 695-696.

⁹¹ Exhibit D24.

⁹² T 170.

Not before? Not before. No.

[59] I do not accept that Mrs Koufos told Dr Shempp and Dr Edwards that she had seen batteries in the area that Manoli played in. The introduction of batteries in her narrative is more likely to be a reconstruction after seeing Dr Kilham's report. The above evidence shows some acceptance of this. Mrs Koufos said nothing of batteries to Mr McClurg who inspected for lead in 2006,⁹³ although she was concerned to identify an area of potential contamination.⁹⁴

[60] Mrs Koufos also gave evidence that in 2007 at Caltex Palmerston she told the then tenancy manager Ramesh Jeffrey that she had told him about all the ashes in October 2005 when he did the inspection and that she had wanted him to have a look. She told him about Environmental Health Reports and that there was lead. She said he did not know anything about that.⁹⁵ In cross examination she said she mentioned batteries.⁹⁶

[61] Ramesh Jeffrey states⁹⁷ that at that time (during the course of the tenancy) tenants would have been advised to remove and clean up rubbish. He states he would not have overlooked such things and a tenant would be required to rectify it. His view of the relevant documents at the time do not record any maintenance issues, although he has cited records about other tenancy issues; the purchase of trees (21 July 2005) and replacement of a hot plate (9

⁹³ T 712.

⁹⁴ T 717.

⁹⁵ T 59-50.

⁹⁶ T 249.

⁹⁷ Exhibit D30, at 32-34.

August 2006). His evidence was he did not recall having any conversation with Mrs Koufos at the Caltex Service Station after his inspection of October 2005.⁹⁸ I conclude it is unlikely that this encounter took place. I cannot rely on the evidence of Mrs Koufos on this point

- [62] According to Dr Duff's first report Mrs Koufos told Dr Duff of the burning of car batteries by previous occupants in the garden of the home. The assessment forming the basis of Dr Duff's first report was not, however, done until 14 August 2008. I do not accept Mrs Koufos' evidence about telling various medical practitioners and others about batteries before Dr Kilham's 2007 report.

The question of a phone call alerting Territory Housing of the presence of batteries and rubbish on the premises

- [63] In support of her evidence that there were batteries in the backyard and that Territory Housing was informed of this, Mrs Koufos told the court that she rang Territory Housing and spoke to someone named "Jan" on 23 June 2005; that she told her there was rubbish in the backyard and asked for someone to clean it up. She said she heard other persons in the background saying 'we don't clean up rubbish'. She said Jan then said to her 'we don't clean up rubbish, this is your responsibility since you have the keys'. Mrs Koufos said she told her there were burnt batteries during this alleged conversation.⁹⁹

⁹⁸ R 249.

⁹⁹ T 28.8.

[64] Ms Saunders’¹⁰⁰ evidence was that she would not have said the words alleged to a tenant. She told the court the Casuarina office was open plan. She did not hear anyone say the alleged words. If she had received such a phone call she would have directed it to the Tenancy Manager. Ms Saunders agreed in cross examination with the proposition it was *possible* that she could have put Mrs Koufos on hold and that the call went to someone else; and it was *possible* that someone else said it was her responsibility.¹⁰¹ In my view however, the probabilities are against the conversation taking place as alleged by Mrs Koufos.

[65] Ms Jan Wray gave evidence that she was the only person called “Jan” in the office at the relevant time. Both Jean Saunders and Jan Wray deny having such a conversation with Mrs Koufos. Both were aware of Territory Housing’s policy and practice at the time and both deny that they would have said this to a tenant. I accept their evidence. It would be extraordinary that Territory Housing would both misplace the Property Condition Report containing the alleged amendment noting batteries or rubbish *and* turn Mrs Koufos away after a phone inquiry allegedly notifying the same. I accept that no management system or messaging system is free from error, including the systems used by Territory Housing. It is possible that calls may be missed or re-directed. Given the weaknesses identified in Mrs Koufos’ evidence, it is likely her evidence on this point is incorrect.

¹⁰⁰ Ms “Jean” Saunders, then allocations officer, Territory Housing.
¹⁰¹ T 29.

Evidence of Batteries and Rubbish at the Premises Prior to 2005

[66] The plaintiff led evidence of sightings of batteries prior to 2005 in support of the contention that Territory Housing was or should have been aware of the presence of batteries, other suspect rubbish, and therefore possible lead contaminants.

[67] Dr Hollingsworth, who lived nearby gave evidence that on two occasions, (that must have been prior to 2004), he saw batteries at the premises. On one occasion when he was chasing his rabbit he saw a car panel and car batteries along the back fence amongst Cassava plants.¹⁰² He said there were several batteries.¹⁰³ He said there was a “burnt sort of car battery sticking up out of the ground” in a depression of 200-300mm deep and one metre wide near the back door.¹⁰⁴ He did not say anything to the then tenant, although he appreciated there may be a risk.¹⁰⁵ This may lend some weight to Mr Koufos’ evidence that he found batteries when he was digging, but that does not mean it was something that could be seen by a reasonable regime of periodic inspection. It is not known how long the batteries he says he saw were present. Dr Hollingsworth’s evidence is a recollection from some eight years before he gave evidence. Although he had an interest in hazards, he was also focussed on chasing his rabbit on at least one of the occasions.¹⁰⁶ This evidence does not in my opinion detract from the conclusion that the probabilities favour the evidence given by Ms Moar and

¹⁰² Marked by him on Exhibit D 6; T 265.

¹⁰³ T 264-266.

¹⁰⁴ T 267.

¹⁰⁵ T 267.

¹⁰⁶ T 267; 266.

the evidence of numerous Territory Housing inspections both before and since that indicate there was no visible presence of batteries or other rubbish.

[68] A Ms Lurdes Pereria also lived in Witherden Street and gave evidence that on one occasion she observed numerous batteries on the premises in the early 1990's, some 12-13 years prior to the plaintiff's occupation of the premises and 20 years before giving evidence before the court.¹⁰⁷ She gives no evidence of seeing batteries on any later occasion. She said the batteries were being melted down to make lead fish sinkers by previous tenants. It is difficult to place significant weight on the accuracy of her observations in these circumstances. Given the material about the inspection regime, I cannot conclude Territory Housing inspections of the premises at that time or since then were not reasonable. I cannot conclude on balance that batteries or other known contaminants would have been seen on reasonable inspection. The premises were inspected many times since the reported observations of Ms Pereria, including close to the time of occupancy by the Koufos family.

[69] I admitted a statement of Mr Peter Hall, (deceased on 10 July 2011), pursuant to s 26D *Evidence Act* (NT). Mr Hall's statement makes reference to parties being held on the premises during which burning was carried out. It was submitted this was possibly during the period Mrs Edwards, (who did not occupy the premises), was the lessee. It was suggested that this burning

¹⁰⁷ T 559.

could have been instigated by strangers or guests of Mrs Edwards in her absence.

[70] I do not give Mr Hall's statement weight (s 26F *Evidence Act* (NT)). The court is not in a position to consider his reliability or credit. His statement would need to be considered against evidence that there were no reports or complaints of anti-social behaviour at the relevant time.¹⁰⁸ The defendant has been unable to cross examine the late Mr Hall. The records indicate Ms Edwards only had keys from 27 May to 6 June 2005.

[71] On behalf of the plaintiff an ingenious submission has been made, drawing on Mr Hall's statement as an explanation for a burnt area in the soil and timber protruding. It was submitted the evidence of a burnt patch and batteries in previous tenancies likely means batteries had sunk into the ground; that no inspection took place at the time Mr Hall observed parties as Ms Edwards did not occupy the premises; that Ms Moar may have overlooked the fire pit which was created for the party but, it was submitted this should not have escaped the observation of the defendant's property managers. The consequence is said to be that the defendant ought to have known of resulting hazards. From the records, Mr Singh told the court Ms Edwards received a refund because she did not move in. In my opinion there is not a sufficient foundation to draw inferences of this significance in the face of evidence to the contrary from Ms Moar and Territory Housing Officers.

¹⁰⁸ Affidavit, Pratap Singh, 26 April 2012, paras 53-54.

[72] The references generally made to there being a “fire pit” do not correspond with the evidence of either Mr or Mrs Koufos as to what they saw in the backyard when they moved into the premises. In my view there was nothing that could reasonably put Territory Housing on notice of a hazard. The regime of inspections was a reasonable one. Mr Singh in particular gave evidence of his practice with inspections and how he followed that practice.

[73] It is the case that in October 2006 when soil testing of the yard took place, Mrs Koufos pointed out two areas in the yard, one in the front yard and one in the back yard where she said the children played. Mrs Koufos had initially suggested at an inspection on 6 October 2006 that the plaintiff’s contact with lead was from eating paint in the premises. At that time Mrs Koufos had said nothing about batteries in the backyard. In terms of what was observed at the inspection arranged at the premises in October 2006, (to inspect for sources of lead exposure), Mr Brian McClurg of Territory Housing inspected a burnt patch of ground in the rear of the premises as well as paint and other possible sources in the house.¹⁰⁹ The burnt patch was an area close to the clothesline,¹¹⁰ the size of a 44 gallon drum. The material he observed, he said, looked like someone may have turned over a Webber type barbecue to clean it out.¹¹¹ The burnt area appeared to be recent; it was darker than the rest of the area and there were small dark

¹⁰⁹ Exhibit D30, Statement of Brian McClurg, 3 November 2008, at 2.

¹¹⁰ T 710.

¹¹¹ T 711.

pieces of possibly charcoal.¹¹² It did not cause him concern from the point of view of being a source of lead.¹¹³ His evidence provides a more detailed description of the ‘burnt patch’ than appears in his statement, but I have no reason to doubt the accuracy of his evidence on this point. Ms Clements also observed an area “quite darker” than the surrounding areas.¹¹⁴

[74] In October 2006 a visibly darker patch of ground was observed close to the clothesline. There was one significant lead level detected in sampling of that patch, but the darkened patch is quite different than what was described by Mr and Mrs Koufos when they moved in. The significance of the lead level sampled from the soil is discussed later in these reasons,¹¹⁵ however in as much as it may be suggested to be evidence supportive of the visible presence of a lead contaminant, in my view the evidence produced from the soil sampling is not capable of supporting an inference that a known contaminant such as batteries or ash or other suspect material was visible during reasonable inspection.

[75] The far more persuasive evidence is the documentary evidence of the Property Condition Report, the previous reports of inspections, the evidence of the immediately previous tenant Ms Moar and of Territory Housing officers who inspected the premises.

¹¹² T 711.

¹¹³ T 712.

¹¹⁴ Exhibit D30, Statement of Natasha Clements, 25 May 2012, and annexure ‘A’.

¹¹⁵ Paras [98] – [110].

The question of whether soil testing and associated steps taken by the defendant were reasonable once the defendant was informed of the plaintiff's blood lead level

- [76] Territory Housing was notified by Mrs Koufos on 22 September 2006 that Manoli had been eating the paint from the house and was sick.¹¹⁶ Natasha Clements, an Environmental Health Officer with the Department of Health and Community Services was notified on 29 September 2006 by a paediatrician¹¹⁷ from the hospital that a child had elevated blood lead levels. On the same date, Ms Clements contacted Territory Housing to advise them of this notification. Dr Shempp's report indicating 'mild lead intoxication' was faxed to Ms Clements on 4 October 2006.¹¹⁸
- [77] I accept it is the position, as submitted on behalf of the plaintiff, that as a public authority, Territory Housing owed a duty to exercise reasonable care in the circumstances to avoid foreseeable risk of injury. Importantly, after this date, Territory Housing had knowledge that there was a potential for lead contamination.¹¹⁹
- [78] The inspection of the premises in response to the report of potential for lead exposure took place on 6 October 2006. Brian McClurg and Wally Jenkins inspected the premises for possible lead sources.¹²⁰ At that inspection Mrs Koufos repeated that the plaintiff was exposed to lead due to eating paint.

¹¹⁶ Exhibit 36 at 400, Tenancy Management System Entry, page 25.

¹¹⁷ Dr Schempp, most likely.

¹¹⁸ Exhibit D 30 at 243.

¹¹⁹ Evidence of Mr McClurg, T 709.

¹²⁰ Exhibit 36, Agreed Tender Book, Annexure 'B' to Statement of Francis Xavier Schobben at 256.

Lead in the house paint was quickly ruled out as the premises were painted well after lead was removed from painting products.

- [79] Soil samples were taken by Ms Clements on 31 October 2006. One of the samples was taken from an area pointed out by Mrs Koufos in the backyard. This was a darkened patch near the hills hoist, shown in the tendered photographs.¹²¹ It was pointed out by Mrs Koufos at the time of the inspection as one of the places that Manoli played. This area does not meet the description of the suspect area of alleged batteries as described in evidence by Mr and Mrs Koufos in 2005 about moving in.
- [80] Mrs Koufos was not informed of the results of the sample testing for seven months after the testing. The soil samples taken by Ms Clements on 31 October 2006 were delivered to Northern Territory Environmental Laboratories Pty Ltd (NTEL) on 31 October 2006.¹²² Ms Clements selected NTEL as they were the only laboratory in the Northern Territory undertaking chemical testing and analysis.¹²³
- [81] In my view the inspection of 6 October 2006 and the taking of soil samples on 31 October 2006 and forwarding them to NTEL were reasonable and timely responses in the circumstances. Between those dates, Ms Clements had also contacted NTEL to ascertain what was required for soil sampling.¹²⁴ By letter attached to the relevant pathology report Dr Schempp said the

¹²¹ Statement of Ms Clements, Exhibit D30, 411-415.

¹²² Exhibit D36, Agreed Tender Book, 281.

¹²³ Exhibit D30 at 411.

¹²⁴ Exhibit D30 at 248.

plaintiff had ‘mild lead intoxication’. At a blood level of .79 $\mu\text{mol/L}$,¹²⁵ according to the same pathology report, the blood lead level was in a category of ‘possible exposure’.¹²⁶ In her letter to Dr Ramdoss (the plaintiff’s GP), Dr Schempp noted aspects of the plaintiff’s development and indicated his symptoms could very likely be due to lead intoxication. She also wrote that Autistic Spectrum Disorder was possible. The main concern she noted was, however his speech development. At age four he was only using single words and mainly gestures.¹²⁷ On the advice of Dr Schempp, the other children in the family were tested for lead. A notation on the Tenancy Management System of 16 October 2006 indicates Mrs Koufos advised Territory Housing that her other children had been tested for lead; it was found that the levels were normal and that she would advise Ms Clements.¹²⁸

[82] NTEL’s report of the soil testing results from the samples taken on 31 October 2006 are dated 7 March 2007. As will be discussed later in these reasons, a sample from the backyard was a level of 449ppm, a level of lead beyond the acceptable public health level that would be expected to trigger an investigation.¹²⁹ It is likely, that because of how busy NTEL were due to their responsibility to respond to water testing requests over the wet season and with staff on leave during the Christmas period, there was some delay. It is likely the defendant received the report between 7-12 March 2007.

¹²⁵ Micromoles/litre.

¹²⁶ Exhibit D36 Agreed Tender Book, 160, Pathology Report, 29 August 2006.

¹²⁷ Exhibit D36, Agreed Tender Book, 706.

¹²⁸ Exhibit D36, Agreed Tender Book at 399.

¹²⁹ Paras [98] – [110].

After that delay of five months there was a delay of about two months between receipt of the report by the defendant and providing it to Mrs Koufos. In my view that delay of two months and the delay overall of some seven months from the time the samples were taken, up until to the results were given to Mrs Koufos was unreasonable.

[83] Mr Schobben, the Director of Environmental Health agreed with the proposition put to him that five months was a very long time to wait for the test results and that Territory Housing could potentially have obtained the results back in a few weeks.¹³⁰ This informs my view the defendant did fail to act reasonably by not obtaining the soil sample test results in a timely manner and providing the results in a timely manner to Mrs Koufos. In terms of addressing the nature and extent of the risk of harm, and what is a reasonable response to it, I bear in mind it is necessary to pay close attention to the degree of risk prospectively rather than retrospectively when considering what is reasonably required by way of response to the risk of injury.¹³¹

[84] In *Wyong Shire Council v Shirt*,¹³² Mason J held that to determine if there has been a breach of the duty of care, it must first be asked whether a reasonable man in the defendant's position would have foreseen that his conduct involved a risk of injury to the plaintiff. If the answer is in the affirmative, it must be determined what a reasonable man would do by way

¹³⁰ T 740-743.

¹³¹ *Roads and Traffic Authority of NSW v Dederer* (2007) 234 CLR 330 at [49].

¹³² (1980) 146 CLR 40, 47-48.

of response to the risk. Relevant to this assessment is a consideration of the magnitude of the risk and the degree of the probability of its occurrences, along with the expense, difficulty and inconvenience of taking alleviating action. A risk of injury that is remote may constitute a foreseeable risk, provided it is not far-fetched and fanciful. The magnitude of the risk and its degree of probability must also be considered with other relevant factors.

[85] While in my view there was nothing to put the defendant on notice at the commencement of the tenancy that anything more than its usual inspection was required, (I accept readily there was no need for expert testing at that time), on being notified of Manoli's blood lead level of .79 $\mu\text{mol/L}$ (16.7 $\mu\text{g/dL}$),¹³³ the discharge of the landlord's duty required Territory Housing to undertake inspection and testing within a reasonable time to ascertain, if possible, whether there was a source of lead and take appropriate remedial steps.

[86] The National Health and Medical Research Council recommends a blood lead level of below 10 $\mu\text{g/dL}$ for all Australians. There is no safe level of lead, however a blood lead level over 10 $\mu\text{g/dL}$ reasonably required a timely response, not only in terms of the inspection and soil sampling which, in my view, was timely, but also to ensure the test results were available in a reasonable time. In terms of the significance of the blood lead level, Dr Schempp's description of "mild lead intoxication" and the attached pathology information indicating it is within the range of "possible

¹³³ Microgram/decilitre.

exposure”, (rather than “possible toxicity” or “lead poisoning”), in the context of a young child in a family home, did require more from Territory Housing to discharge its duty. In my view, while not indicating a toxic level of lead, the trigger level of 10 µg/dL exists as an indicator of the need for investigation. In these circumstances, in my opinion, obtaining the results of soil sampling in a timely fashion was required to discharge the duty. While the risk of harm at 16.7 µg/dL was not in itself indicative of toxicity, given the possibility of continued exposure and therefore further raised blood lead levels, in my view there was a foreseeable, albeit remote, risk to a tenant. As Dr Kilham, an expert in these matters stated, ‘if you find a level of 10 or above you should look for where the abnormal source of lead is coming from’.

[87] In the circumstances of a public authority, even though there was no direct control of NTEL by the defendant, more steps should have been taken to impress on NTEL the importance of obtaining the results. Alternatively, attempts to obtain testing elsewhere when it became apparent NTEL were unable to provide a sample in a reasonable time, ought to have been made. That is not however, the end of the matter.

[88] The evidence when taken as a whole indicates that this shortcoming in timeliness by the defendant did not lead to the plaintiff being exposed to lead; or further exposed, and did not contribute to the conditions he suffers from.

- [89] The plaintiff's case is that the defendant failed to inform Mrs Koufos that she should prevent her children from playing in the 'fire pit'. For reasons already discussed, I do not accept there was a 'fire pit', but I accept there was an area where Manoli played, pointed out by Mrs Koufos during the investigation when soil samples were taken.
- [90] Ms Clements stated she does not now recall making any recommendations to Mrs Koufos regarding access to or use by the children or family members of the suspect area from where the soil samples were taken.¹³⁴ She stated however, that in accordance with the usual investigative procedure she followed at the time, she would have told the plaintiff's mother when she attended the premises that she should not allow her child to eat non-food substances such as paint, ash, dirt or soil.
- [91] I give little weight to the evidence of Mr Schobben that Ms Clements told Mrs Koufos at the inspection that she should prevent the plaintiff having access to the area.¹³⁵ Not because I think it is untruthful, but it is not capable of being tested and goes further than Ms Clements' own statement. I accept however, that this type of advice is the advice that would be expected to be given by an environmental health officer and in any event was common sense not to allow a child to play in a suspect area.
- [92] Although Mrs Koufos' evidence in chief was that she did not prevent the plaintiff playing in the area while awaiting the soil testing results, (because,

¹³⁴ Exhibit D30 at 412.

¹³⁵ T 740.

she said, she was not advised to do so, and hence Manoli continued to play in the area), in cross examination she acknowledged that she was aware at that time that the area could have been a source of the plaintiff's lead intoxication; she was worried about his health and did her best to keep him away from the area - but he continued to play there "as I was still not aware of any of the reports coming back to me as to where that source of lead had come from".¹³⁶ I do not consider this to be a credible response by her in the circumstances. On balance, in my view sufficient information was communicated to Mrs Koufos to convey to her that she should not allow the children to play in the suspect area. If I am wrong about that, she acknowledged she knew in any event not to allow it.

[93] Of significance is the fall in the plaintiff's blood lead levels from .79 $\mu\text{mol/L}$ (16.7 $\mu\text{g/dL}$) after October 2006. This is consistent with being given advice or at the very least an awareness of the need for Manoli to stay away from the suspect area. The subsequent lead levels were:

6 November 2006	.37 $\mu\text{mol/L}$ (7.4 $\mu\text{g/dL}$)
1 December 2006	.36 $\mu\text{mol/L}$ (7.2 $\mu\text{g/dL}$)
28 December 2006	.28 $\mu\text{mol/L}$ (5.6 $\mu\text{g/dL}$)
28 February 2007	.21 $\mu\text{mol/L}$ (4.2 $\mu\text{g/dL}$)

¹³⁶ T 226-227.

[94] It is also telling that Mrs Koufos was offered both alternative accommodation and excavation and remediation of the soil in the backyard. Mr McClurg said that in May 2007, there was a conversation about the possibility of relocating the Koufos family.¹³⁷ It did not happen because Mrs Koufos did not want to move.¹³⁸ Xavier Schobben discussed the matter with an officer from Territory Housing about relocation or remediation in May 2007. Despite there being only one sample of concern tested, a precautionary approach was taken and an offer was made to either relocate or to have the site remediated and re-sampled.¹³⁹ Mr Schobben was subsequently informed that the tenant had elected not to move or to have the soil remediated.¹⁴⁰

[95] Mrs Koufos said that on the advice of her solicitor she decided to stay in the house. She was concerned about further injury that might have been sustained by Manoli staying in the house, but she stayed because she had nowhere to go. She also said she did not accept the offer “because the house was being currently under investigations”.¹⁴¹ I do not accept Mrs Koufos’ evidence about being unable to leave. She obviously enjoyed living in the house; the house is in a particularly good area for her.¹⁴² She believed the concerns she had previously held for her children in relation to lead, had

¹³⁷ T 714.

¹³⁸ T 714.

¹³⁹ T 738.

¹⁴⁰ T 738.

¹⁴¹ T 48.

¹⁴² T 252; Evidence of Michael Mailis, T 401.

passed. At the time of trial, she and the children were still living in the house.

The source of lead causing the plaintiff's elevated blood lead level

- [96] While I appreciate from the evidence given that caution must be applied to relying on the results of a single elevated soil sample, in my view the probabilities favour a conclusion that the soil in the backyard of the premises contributed to the plaintiff's elevated blood lead level (.79 μ mol/L; 16.7 μ g/dl). I accept there is no proven correlation between the level of lead in the soil and blood lead levels.
- [97] The blood lead levels of Manoli's siblings were not of concern. Their blood lead levels in October 2006 were (Nikolas) .07 μ mol/L; (Aphrodite) .10 μ mol/L; (Pantelys) .22 μ mo/L; (Panayotis) .11 μ mol/L. It is likely they did not play in the backyard as often as Manoli.
- [98] The soil sample taken in October 2006 from the backyard showed the presence of lead in the amount of 449ppm. This was from the area Mrs Koufos pointed out. That Manoli played there regularly was not challenged. It is reasonable to accept Manoli's toys were modern and unlikely to contain lead. The measure of 449ppm was significantly higher than the (Australian) National Environmental Protection Council's health investigation level of 300ppm.¹⁴³

¹⁴³ Evidence of Xavier Schobben; Exhibit D 30 at 251, 259, Health – Based Soil Investigation Levels.

[99] Such a level of lead in the soil does not however indicate toxicity or necessarily that it is harmful. A 1995 study revealed that 40 percent of background soil samples from residential areas in Brisbane and Sydney exceed the trigger threshold of 300ppm.¹⁴⁴ They are the levels at which an investigation or inquiry might commence.¹⁴⁵

[100] The evidence overall indicates the prevalence of lead in the community has fallen significantly over the last (approximately) 30 years. Upon inspection of the premises for lead, Mr McClurg noted a mag wheel which appeared to have lead weights in it. Manoli was seen sitting in the wheel at the time. The wheel was sitting alone with another stack of about five. It had a lead weight in it.¹⁴⁶ This was not tested, however Mr McClurg is a trained eye for noting ordinary objects that contain or are associated with lead. While it is possible there may have been other sources of lead, in my view the probabilities favour the backyard as the source of lead and the main contribution to Manoli's blood lead levels. Dr Kilham's evidence was that the soil was the most likely source.

[101] Such a conclusion is consistent with Manoli's blood lead levels falling after he was stopped from playing in the area. As I have already found, Territory Housing were on notice after receiving the results of Manoli's lead levels, that there may be a source of lead in the premises. Those levels reduced

¹⁴⁴ Exhibit D33, Xavier Schobben at 733.

¹⁴⁵ Evidence of Xavier Schobben at 735 and Exhibit D30 at 261-262.

¹⁴⁶ T 712.

because of advice given by Ms Clements or by Manoli's parents exercising common sense by not allowing him to play in the suspect area.

The level of lead in the soil in 2005

[102] As is discussed later in these reasons, to prove the most serious of his symptoms and conditions were caused by lead exposure, it must be proven the plaintiff's blood lead levels were much higher than .79 $\mu\text{mol/L}$ (16.7 $\mu\text{g/dL}$). The preponderance of expert evidence indicates a much higher level of lead would be required to produce the symptoms and conditions Manoli suffers from. In addressing this issue, the plaintiff submits it is probable that the lead levels in the soil were much higher in 2005 when the plaintiff moved into the premises than in October 2006, (when the soil testing took place), or May 2007, (when the results were known). Such a finding would be in keeping with the theory that the plaintiff's blood lead levels would also have been higher at an earlier time, thus capable of causing the plaintiff's symptoms and conditions.

[103] The evidence does not in my view support a finding that there were higher levels of lead in the soil in June 2005. The plaintiff relies on the wet season of 2005 and suggests that there would have been soluble elements of lead which would be washed away or that particles of lead would move if the soil moved in circumstances of heavy rain. Further, it is suggested that the wet season of 2006-2007 resulted in all subsequent soil lead levels being vastly lower than 449ppm. (In May 2007, the suspect area was tested and produced

a result of 26ppm; 15 June 2007 it was 9.3ppm; 4 February 2008 it was 21.6ppm).

[104] Mr Schobben agreed that material may be moved around in wet seasons and soil conditions may change from wet season to wet season.¹⁴⁷ He said however that it was speculation to conclude there was a higher soil reading in 2005. He explained that lead, as a heavy metal, differed from bacteria and he would not expect it to be washed across the yard in the wet season.¹⁴⁸ Although Mr Harrison from Above Capricorn Technologies said that lead is enclosed in soil and if the soil moves it would be expected material within the soil would move, he was prepared only to say that lead levels may have been higher at an earlier time. In his written report he stated it was not valid to draw conclusions about prior lead values.¹⁴⁹ It is possible that soil moved at the premises in the previous wet seasons but there is no direct evidence about the movement, or “wash through” of soil from the premises in any of the relevant wet seasons.

[105] Other experts could not take the matter much further. Professor Winder, (an occupational toxicologist), acknowledged he was not a soil expert¹⁵⁰ but assumed that rain may affect the distribution of lead in soil depending on run-off and the extent of the rain. He took the view based on common sense that if there had been some “wash through” then lead levels in the soil may diminish over time.

¹⁴⁷ T 739.

¹⁴⁸ T 744, 746.

¹⁴⁹ Exhibit P10 at 2; T 308.

¹⁵⁰ T 490.

[106] Although not an opinion within his area of expertise, which he readily acknowledged, Professor Winder's view, I agree is reflective of a deal of common sense and experience, however, without some further evidence about whether soil from this backyard tended to wash away in wet seasons, it is not possible to draw the inference sought on behalf of the plaintiff.

[107] It is unknown how or if the soil moved around and to what extent, if it did. It is an ordinary backyard of a family home. A range of issues of a physical nature may cause a change in the concentration of the initial source. The evidence is not firm enough to come to a conclusion that on balance, the wet season affected the distribution of lead in the soil. It does not prove there was a higher concentration or distribution of lead, at an earlier time.¹⁵¹ To conclude otherwise would be impermissible speculation.

Whether the soil sample of 449 ppm was representative

[108] The defendant argues the soil sample of 449ppm was aberrant or non representative. On whether the backyard sample was aberrant, it is recognised that Ms Clements was not trained in taking soil samples; skill and training are required otherwise an aberrant, non representative sample may be obtained.¹⁵² It is also the case that no other soil sample taken since that sample has been anywhere near that range. Professor Winder agreed that very little can be made out of one elevated soil sample.¹⁵³ The direct evidence about this is equivocal, however, taking the sample into account

¹⁵¹ Professor Winder, T 516.

¹⁵² T 736, 310-311.

¹⁵³ T 502.

with all of the other evidence, it can safely be concluded the sample was not aberrant.

[109] In coming to this conclusion I am influenced by Dr Kilham's evidence to the effect that although there is no proof that Manoli was exposed to lead in the backyard, the soil was the most likely cause of any lead contamination Manoli suffered. He said the evidence available to him was that Manoli was exposed to a small amount of lead and this was reflected by a fairly low blood lead level.¹⁵⁴ Dr Kilham also said that if Manoli had been seriously poisoned with lead from soil in a pit, the soil sample would have been in the thousands, not 449ppm.

[110] Although it remains a possibility that the backyard sample is aberrant, after taking into account all of the known evidence, I conclude that for some of the time Manoli played in the yard he was exposed to a level of lead found to be 449ppm. It is not known how long he was exposed to that lead source or how he may have ingested the lead, (save that consistent with his condition, pica may be involved) but, on the balance, soil in the backyard was the main contribution to his elevated blood lead level.

The plaintiff's blood lead levels prior to the testing of 29 August 2006

[111] As has been discussed, the testing of 29 August 2006 revealed the plaintiff's blood lead level was .79 $\mu\text{mol/L}$ ($\mu\text{mol/L}$ "micromoles per litre"); 16.7 $\mu\text{g/dL}$ ("micrograms per decilitre"), exceeding the reference range

¹⁵⁴ T 616.

indication of 10 µg/dL. That was Manoli's highest recorded blood lead level. The plaintiff's case is that his blood lead level was at a much higher level prior to the testing of 29 August 2006.

[112] The plaintiff called Dr Duff, a psychologist, neuroscientist and clinical nutritionist who holds, with other qualifications, a doctorate in clinical neuroscience. Dr Duff's opinion¹⁵⁵ was that the lead exposure would have been at its highest in June 2005. He said that blood lead levels indicate recent exposure to lead; and that a blood lead level will reduce over time. It follows that if a test was done a year after he was exposed to lead there would have been a significantly reduced result.¹⁵⁶ Based on a paper from the (American) Centre for Disease Control (CDC) which found that the half-life clearance of lead from the body is estimated at one month, and applying the same formula retrospectively, according to Dr Duff, Manoli's blood lead level in November 2005 would have been 100 µg/dL.¹⁵⁷ He said this was a conservative measure and was consistent with his symptoms. This reasoning is highly contested by the medical experts called by the defendant.

[113] Dr Duff said it was possible that the blood lead level was lower than 100 µg/dL, suggesting 70 or 60 µg/dL; that it would have peaked at around November 2005 but that does not necessarily assume a single dose at an earlier time; it may come from a single dose but that was unlikely. He said

¹⁵⁵ Exhibit P12; Exhibit P13.

¹⁵⁶ T 435-436.

¹⁵⁷ T 437.

it may have peaked by accumulation from July to November¹⁵⁸ from continuous exposure.

[114] As there was a forward depletion of lead, his view was that logically the same should apply retrospectively. He considered it a matter of mathematics to apply the formula retrospectively. Commenting on the NHMRC paper,¹⁵⁹ referring to blood lead levels being the bio-marker of choice for determining exposure for the preceding five to six weeks, he said that was a paper for the public to make them aware of what should be done if there is a need for testing.¹⁶⁰ He conceded there was no back-depletion rate consistent with this formula set out in the CDC literature.

[115] Dr Duff agreed he did not have evidence of what happened earlier in time except that Manoli was playing in the dirt probably loaded with lead. That assumption was based on there being batteries burnt by previous tenants.¹⁶¹ He also agreed that absent earlier blood lead levels, it is left to speculation on whether and to what extent there is blood lead levels in Manoli, adding “its speculation based on some science, namely the rate at which lead is excreted from the body”.¹⁶² He also agreed the calculation was an attempt to explain Manoli’s symptoms and his blood lead levels, because he could not assign his symptoms to autism.¹⁶³ Dr Duff denied that death would be the result for many children with a blood lead level of 100 µg/dL; he said at 100

¹⁵⁸ T 448.

¹⁵⁹ Exhibit D 14.

¹⁶⁰ T 451.

¹⁶¹ T 450.

¹⁶² T 453.

¹⁶³ T 453.

there was encephalopathy and anaemia. (This opinion is contrary to the other evidence on the point). He conceded the level may not have been that high.¹⁶⁴

[116] Professor Winder, an occupational toxicologist gave evidence that it was very unlikely that the blood lead level of 16.7 µg/dL was the maximum level. This was based on Manoli having played in the yard for an extensive period; and that he was likely to have engaged in hand to mouth activity. He also concluded that working backwards on the basis of the half life of lead, the levels may have been as high as 100 µg/dL. He indicated this was based on standard toxicological theory of biotransformation and disposition.¹⁶⁵ He said putting a number on it is speculation, but 100 is not unreasonable.¹⁶⁶ In relation to a graph he used to illustrate Manoli's blood levels, Professor Winder said there "is a certain amount of science in this. Not much but some".¹⁶⁷ In contrast with Dr Duff, Professor Winder agreed that at levels in excess of 100 or 150 µg/dL, many children would be dead.¹⁶⁸ He conceded he did not know what Manoli's previous blood level was. He said he thought the previous blood level was much higher.¹⁶⁹

[117] Professor Winder's report commenced with a considerable critique of what he believed the defendant had done or omitted to do without being in possession of the relevant facts. I am not confident Professor Winder has

¹⁶⁴ T 453.

¹⁶⁵ T 532.

¹⁶⁶ T 484.

¹⁶⁷ T 505.

¹⁶⁸ T 505.

¹⁶⁹ T 495

considered his opinion and evidence from a broad or balanced perspective of the different possible factual scenarios prior to the blood lead level of 16.7 µg/dL being taken. I am not satisfied the backward calculations made by Dr Duff and Professor Winder are scientifically sound. I am not prepared to act on them. I found the evidence of Dr Kilham and Dr Marraffa far more persuasive and grounded in the available materials, histories and reports and did not involve speculation of the type engaged in to some degree by Dr Duff and Professor Winder.

[118] Dr Kilham is a paediatrician of long standing who has worked extensively in the area of poisons. He previously had responsibility for the Poisons Centre in New South Wales, then the National Poisons Centre. He has been involved in lead cases since the late 1960's, both with the treatment of lead poisoning and reviewing the literature. He is a member of the National Health and Medical Research Council. Dr Kilham regarded Professor Winder's back calculation as "purely imaginative" without scientific validity.¹⁷⁰

[119] In cross examination Dr Kilham disagreed that he had assumed immediately that at no point had Manoli been exposed to a higher level of lead.¹⁷¹ He said he would be very surprised if Manoli's blood level at the end of 2005 was 60 µg/dL because his symptom pattern was different from what would be expected. At that level he would have expected him to have slowed right

¹⁷⁰ T 598.

¹⁷¹ T 616.

down, be apathetic, constipated all the time and pale. He would have had much higher levels of exposure to produce the noted symptoms. Any *sudden* change in symptoms, he said, would probably not be because of lead. In terms of symptoms of constipation, abdominal pain, vomiting, irritability manifesting as running into walls, he said a person exhibiting such symptoms *may* have been exposed to lead at levels of 40 – 60 µg/dL. He said it would probably be one in 50,000 children showing those symptoms due to lead.¹⁷²

[120] Dr Kilham said that based on the half life of lead theory alone, not on any of the other facts, it is within the bounds of possibility that earlier his blood lead level was 50 – 60; equally, he said the blood lead level could have been half the level of 16.7. He agreed that even if Manoli had steady exposure over time, it is possible that his blood lead level was at one time 60 – 80 µg/dL, but he said this was extremely unlikely. He commented that anything is possible, just speculation, but possible.¹⁷³

[121] Dr Kilham's expert opinion was that Manoli did not present at any time with clear symptoms of lead poisoning or encephalopathy. His blood lead levels were not high enough, and it could not be shown that he had been exposed to lead long enough.¹⁷⁴

¹⁷² T 636.

¹⁷³ T 642 – 644.

¹⁷⁴ T 596.

[122] Dr Marraffa, the consultant paediatrician,¹⁷⁵ called on behalf of the defendant, said Professor Winder's back calculation graph is irrelevant and speculation; that there is no way of proving one way or the other what Manoli's blood lead levels were before August 2006.¹⁷⁶

[123] I do not accept the plaintiff's submission that Dr Kilham sought to take over the role of adjudicator in the case, anxious to impart a point of view. Dr Kilham made a number of concessions in terms of possibilities that were capable of assisting the plaintiff's case; nor do I consider Dr Marraffa had an "agenda". She came to a view early in her involvement in the case, (when consulting for the plaintiff's former solicitor), that lead was not implicated, but she made her conclusions on grounds transparently explained – that developmental problems commenced well before Manoli was exposed to lead¹⁷⁷ and that the level of lead was not in the range of significance.

[124] I consider Dr Kilham and Dr Marraffa have demonstrably superior expertise than Dr Duff and Professor Winder in relation to the question of lead and its affect on children. Dr Marraffa is clearly a leading expert on autism and developmental disabilities. I accept Dr Kilham's and Dr Marraffa's opinions are soundly and scientifically based.

¹⁷⁵ Dr Marraffa is the Deputy Director, Developmental Medicine and Paediatrician to the Autism Assessment Team, Integrated Mental Health Service of the Royal Children's Hospital in Victoria. She serves on the Royal Australasian College of Physicians, Victoria, Committee and worked as a clinician with children with developmental disabilities for over 24 years.

¹⁷⁶ T 817.

¹⁷⁷ T 852.

[125] I do not accept the evidence permits a finding that Manoli's blood lead levels were higher, but if they were, they could not be shown to be at the levels calculated or assumed by Dr Duff or Professor Winder. In my view such conclusions require significant speculation beyond what is permissible. I have considered also the report of Dr Michael Robertson,¹⁷⁸ forensic toxicologist noting the possibility of the extrapolations conducted by Professor Winder, however given the many assumptions required to make those extrapolations, he considers it is a speculative exercise. A similar critique is made by him of Dr Duff's calculations to achieve the peak level he determined.

Observations by witnesses and relevant records about Manoli's physical health

[126] It is common ground that the effect of blood lead concentrations may vary between individuals, children likely being more susceptible, and at certain levels of concentration, lead may produce physical symptoms such as headaches, abdominal pain, constipation, nausea and vomiting, anaemia, fatigue, stupor, slurred speech, numbness and tingling in the extremities. Diarrhoea is less associated with lead. A significant amount of evidence is before the Court about Manoli's physical symptoms.

[127] In general, Mrs Koufos assured the court she had no trouble recalling the various events of 2005 and those prior to 2005.¹⁷⁹ In relation to Manoli's physical health, particularly episodes of vomiting and diarrhoea, she agreed

¹⁷⁸ Exhibit D30, 53-58.
¹⁷⁹ T 63.

she could not recall dates; her memory was not wrong but she “cannot recall the years and the dates and the times”.¹⁸⁰ No parent and not Mrs Koufos could be criticised for not recalling such details. Although ordinary experience would indicate there would be such difficulties with memory, Mrs Koufos persisted with evidence that her memory was correct in relation to the symptoms she observed both before and after the move to the premises. Her evidence was essentially that the vomiting, diarrhoea and constipation did not start until later in 2005; that any previous sicknesses were not of significant concern to her, they were just “flus”.¹⁸¹ Given Mrs Koufos’ unreliability that I have previously discussed, it is primarily other evidence that must be assessed to form reasonable conclusions about Manoli’s physical health.

[128] The lay witnesses gave evidence generally consistent with the plaintiff’s assertion that until after the move to 35 Witherden Street, the plaintiff suffered no particular health problems aside one sign of developmental delay – a speech delay. The impression sought to be conveyed was that Manoli was in good health until the move. The objective medical material contradicts this evidence. I will summarise some of the relevant medical material and Mrs Koufos’ response to the records when they were drawn to her attention.

¹⁸⁰ T 184-185.

¹⁸¹ T 66.

[129] On 11 July 2002, (within 6 days of Manoli's birth), Mrs Koufos took Manoli to the Community Care Clinic. A nurse noted problems with breathing; colour red, crying, pulse 150, very mucousy. He was referred to Accident and Emergency for assessment.¹⁸² Mrs Koufos agreed this incident occurred and that she had taken Manoli to hospital.¹⁸³ The records also indicate Manoli was admitted for observation following a choking episode.¹⁸⁴ Mrs Koufos agreed he was admitted to hospital overnight. Mrs Koufos said at first he did not vomit. She then said she could not recall him vomiting. The medical notes indicate "vomit".

[130] Mrs Koufos was asked whether she could recall that between 11 July and 22 October 2002 she took Manoli to see Dr Ramdoss on seven occasions and to the Community Care Clinic on one occasion. She said she could not recall.¹⁸⁵ She agreed that on 25 November 2002 she took Manoli to the Community Care Clinic. She advised them Manoli had diarrhoea for about one week. She said she was concerned and the clinic spoke to her about the management of the condition.¹⁸⁶ She was asked about further visits to Dr Ramdoss in 2002 and 2003 and she said she took Manoli to him for various problems such as asthma, colds and bronchitis.¹⁸⁷ She could not recall any discussion about a milk allergy. Dr Ramdoss's notes for 20 December 2002 contain a question mark next to the words "milk allergy".

¹⁸² Exhibit 36, Agreed Tender Book at 613, 638.

¹⁸³ T 67-68.

¹⁸⁴ Exhibit 36, Agreed Tender Book at 408-409.

¹⁸⁵ Exhibit 36, Agreed Tender Book at 712, Dr Ramdoss's notes indicate these multiple visits concerned coughs and bronchitis.

¹⁸⁶ T 71; Exhibit 36, Agreed Tender Book at 615.

¹⁸⁷ T 72; Exhibit 36, Agreed Tender Book at 712-713.

[131] Mrs Koufos said she recalled a gastroenteritis episode on 24 November 2002 when Manoli was vomiting and had problems with his bowels but she could not recall discussing dehydration.¹⁸⁸ In relation to the occasions that Manoli was taken to Dr Ramdoss and the hospital between November 2002 and October 2003, (on around a dozen occasions), Mrs Koufos said she was not worried about those incidents. She said it was “for flus”; he would only vomit for one day and the next day he would stop.¹⁸⁹

[132] Mrs Koufos agreed she took Manoli to the Community Care Clinic when she had returned from a visit to Greece and told them Manoli had constant chest infections.¹⁹⁰ She agreed that on 24 November 2003, Manoli’s hearing was tested.¹⁹¹ She agreed she took Manoli to Dr Ramdoss for bronchitis on 12 March 2004.¹⁹²

[133] Mrs Koufos told the court she did not recall taking Manoli to the emergency department on 20 March 2004 for diarrhoea and vomiting.¹⁹³ The relevant medical notes clearly indicate diarrhoea and vomiting; the notes indicate three days of diarrhoea and four days of vomiting. She agreed that this would have been very concerning. She did not recall taking Manoli to the emergency department the following day with vomiting; she could not recall

¹⁸⁸ T 72.5, Mr Palamedes Koufos could not recall Manoli going to hospital with diarrhoea in November 2002.

¹⁸⁹ T 72-73. Exhibit 36, Agreed Tender Book at 713-715; 460-462.

¹⁹⁰ T 73; Exhibit 36, Agreed Tender Book at 615.

¹⁹¹ T 73; Exhibit 36, Agreed Tender Book at 615.

¹⁹² T 73; Exhibit 36, Agreed Tender Book at 615.

¹⁹³ T 72; Exhibit 36, Agreed Tender Book at 451-453.

being told to keep his fluids up.¹⁹⁴ Although Mrs Koufos agreed with the likelihood of taking Manoli to see Dr Ramdoss in May and June 2004, she did not recall a visit on 5 June 2004 when Dr Ramdos discussed the possibility of renal failure.¹⁹⁵ Mrs Koufos said she remembered taking Manoli to the emergency department on 12 August 2004 because of vomiting and diarrhoea for a couple of days.¹⁹⁶ She said she did not recall him being too distressed to be examined. The notes indicate the contrary. She said the whole family were getting sick with flu including diarrhoea and vomiting. She agreed that Manoli having flu with vomiting and diarrhoea was something that happened on a regular basis.¹⁹⁷

[134] Mrs Koufos gave evidence of Manoli having had three falls on his head.

The first was when he was four months old. His siblings jumped on the bed he was sleeping on; she heard a big bang and found him on the bed crying; she took him to hospital; he did not have a lump on his head.¹⁹⁸ Mrs Koufos took him to the emergency department on 22 October 2002. He was not admitted to hospital.¹⁹⁹ The second fall was described as occurring when Aphrodite accidentally dropped Manoli on the floor. Mrs Koufos took him to hospital. She said Manoli had no problems arising from those falls. In cross examination she agreed that on 3 November 2002 she rushed him to hospital because he had been thrown off of the bed by one of the children.

¹⁹⁴ T 74; Exhibit 36, Agreed Tender Book at 449-450.

¹⁹⁵ T 75; Exhibit 36, Agreed Tender Book at 717.

¹⁹⁶ T 75-77; Exhibit 36, Agreed Tender Book at 445-448.

¹⁹⁷ T 77.

¹⁹⁸ T 61.

¹⁹⁹ T 69.

[135] The medical notes indicate this was the second presentation within two weeks when a sibling tried to pick him up and dropped him.²⁰⁰ Mrs Koufos denied that this was second time he had been dropped by a sibling. The note indicates a fall from bed “today”; onto the floor; well today; feeding well; alert and playful; when he woke up, fully responsive. A red area was noted on his forehead. Mrs Koufos said she did not recall that. Mrs Koufos said she did not recall taking Manoli to the emergency department of the hospital on 13 June 2005 for a cut above his eye.²⁰¹ She said she could recall him having stitches in 2011 but not in 2005. She said she did not recall him screaming, refusing to stand up to be examined, or urinating himself when in the emergency department. She did not recall taking him home on that occasion. The notes of that examination on 13 June 2005 record such a presentation.²⁰²

[136] The impression Mrs Koufos and other witnesses attempted to convey to the court that aside from “flus”, Manoli did not suffer from symptoms of vomiting and diarrhoea until after the move to the premises, is not borne out by the objective material. The vomiting and diarrhoea, (the latter of which Mrs Koufos thought was suggestive of lead poisoning but is not generally associated, and effectively suggested coincidence with the move to the premises or soon after), is contrary to the objective evidence. Although it is accepted it would be difficult to remember all of the times a young child

²⁰⁰ Exhibit 36, Agreed Tender Book at 465.

²⁰¹ T 77-78.

²⁰² Exhibit 36, Agreed Tender Book at 444.

would be taken for medical treatment; to go further and positively assert this coincidental change in the physical condition, in my view diminishes the credibility of the plaintiff's case.

[137] There are seven clear medical notations of vomiting, diarrhoea or gastroenteritis before June 2005.²⁰³

[138] In terms of these particular symptoms after June 2005, the plaintiff was seen on 22 October 2005;²⁰⁴ 17 December 2005;²⁰⁵ 28 February 2007;²⁰⁶ 3 March 2007;²⁰⁷ 1 January 2011.²⁰⁸ If anything, those particular symptoms settled. In November 2006, additionally, he was treated for constipation.²⁰⁹

Although constipation may be associated with lead toxicity it may also be due to a range of causes. Diet and treatment for anaemia were at times suspected in the case of Manoli. Although Mrs Koufos gave evidence of Manoli developing constipation interspersed with bouts of green-coloured diarrhoea after the move, this is not evident in the medical records relevant after the move. It was however the history she gave a number at a later time to Dr Duff and Dr Marraffa.

[139] The other witnesses who gave evidence about the alleged change in symptoms cannot be regarded as reliable. Mr Koufos gave evidence that

²⁰³ 24 November 2002, Dr Ramdoss; 25 November 2002, Community Care Clinic; 28 July 2003, Dr Ramdoss; 2 October 2003, RDH Emergency; 20 March 2004, RDH Emergency; 21 March 2004, RDH Emergency; 12 August 2004, RDH Emergency.

²⁰⁴ Dr Ramdoss.

²⁰⁵ RDH Emergency.

²⁰⁶ Dr Ramdoss.

²⁰⁷ Dr Ramdoss.

²⁰⁸ Dr Ramdoss.

²⁰⁹ 23 November 2006, RDH Emergency.

before moving to Witherden Street, Manoli's health problems requiring a doctor were "one or two times when he was bitten by a dog and then a couple more times when he just fell down and got himself hurt".²¹⁰ In terms of vomiting and diarrhoea he said "Maybe he got sick, I don't know too much because I was at work". He acknowledged one occasion when Manoli was taken to the doctor.²¹¹ When put to him specifically, he could not recall Manoli going to hospital with diarrhoea in November 2002;²¹² March 2004²¹³ or August 2004.²¹⁴

[140] Olga Fountis was asked about Manoli going to hospital many times before moving into the premises. She said at first that this only occurred once they had moved; she agreed that before moving to Witherden Street, Manoli had gone to hospital because "maybe he had an accident";²¹⁵ she denied he had to go to hospital before moving to the premises for vomiting or diarrhoea. Mrs Fountis' preparedness to tailor her evidence to suit what she most likely believed was beneficial to the plaintiff's case diminishes her credit.

[141] In terms of physical ailments after moving to the premises, Manoli was seen for an abscess in the mouth (gingivitis);²¹⁶ distress and screaming after a fall from shopping trolley;²¹⁷ on 22 October 2005, seen by Dr Ramdoss for

²¹⁰ T at 277.

²¹¹ T at 281.

²¹² T at 281.

²¹³ T at 281.

²¹⁴ T at 281.

²¹⁵ T at 363.

²¹⁶ 10 July 2005, RDH Emergency notes, Exhibit 36 Agreed Tender Book at 441-443.

²¹⁷ 2 September 2005, RDH Emergency notes Exhibit 36 Agreed Tender Book at 439-440.

diarrhoea;²¹⁸ 17 December 2005, seen for vomiting and diarrhoea;²¹⁹ 17 February 2006, seen in RDH for an attack by a dog; throughout June and July 2006 seen by Dr Ramdoss on four occasions for bronchitis or tonsillitis.

[142] Manoli was admitted to RDH on 23 November 2006 with severe constipation; on 28 February 2007 he was seen by Dr Ramdoss for diarrhoea, vomiting, gastroenteritis; some stool samples were taken to test for Rota virus, and Giardia and other conditions. On 3 March 2007 he was seen by Dr Ramdoss again with similar symptoms. On 1 May 2007 he was seen by Dr Ramdoss for a cough; on 1 August and 11 December 2007 he was seen by Dr Ramdoss for tonsillitis. He was seen on 7 March 2008 by Dr Ramdoss for gastroenteritis; and in April 2008 for bronchitis and iron deficiency. On 15 November 2008 he was seen for recurrent urticarial rash; on 14 January 2009 for fevers; in February, March and September 2009 he was seen for a throat infection or fevers. On 1 January 2011 he was seen by Dr Ramdoss for gastroenteritis and Dr Tomlinson for a sore throat and cough. In late 2009 liver function tests were ordered and worsening liver function was noted.

[143] Overall, after moving into the premises and allowing for some period after then, (given the need to bear in mind that symptoms of lead poisoning are expected to be evident gradually after exposure), I am unable to find an increase in the physical symptoms that Manoli suffered. These conditions

²¹⁸ Exhibit 36 Agreed Tender Book at 700.

²¹⁹ RDH Emergency notes, Exhibit 36 Agreed Tender Book at 436-437.

have all been shown to be similar to the childhood conditions he suffered before being exposed to lead. I am not persuaded to the contrary by the assertion that other members of the family were also ill in 2005 but all recovered except for Manoli.

Discussion of whether the condition of anaemia was caused by lead

[144] Both the plaintiff and his siblings suffered anaemia. The blood tests however revealed “microcytic” and “hypochromic” features, consistent with anaemia caused by poor diet. Diet appeared to be of significant concern to Dr Schempp.²²⁰ Dr Schempp recorded a history of Manoli drinking excessive amounts of milk. She commenced ‘aggressive oral iron therapy’.²²¹

[145] Dr Duff believed that lead caused the iron deficiency,²²² however he also agreed that if the siblings had iron deficiency there may be a dietary explanation.²²³ He appeared to have relied on a history, also given in evidence by Mrs Koufos, that ordinary family meals were blended and Manoli would eat those. That may or may not be reliable; what also must be considered is the history of drinking excessive amounts of milk.

[146] Professor Winder told the court he was not surprised at Manoli’s anaemia. His graph suggested anaemia occurs at higher levels of lead (50 – 100

²²⁰ Dr Schempp was not called; however her initial report to Dr Ramdoss, Manoli’s GP, was a significant part of the evidence. See Exhibit 36, Agreed Tender Book at 272-274.

²²¹ Report Dr Schempp, 4 October 2006.

²²² T 435 – 444.

²²³ T 464.

µg/dL).²²⁴ He did not consider it relevant to obtain the medical records regarding the type of anaemia, nor did he consider the relevance of “microcytic” and “hypochromic”. He agreed there may be a common dietary issue amongst the children in the family; he also agreed dietary induced anaemia can affect the behaviour of the child.

[147] Dr Kilham told the Court Manoli had iron deficiency anaemia, quite distinguishable from lead typical anaemia. It tended against a conclusion of lead poisoning.²²⁵ He said Manoli’s iron deficiency anaemia was related to his poor diet from drinking cow’s milk, which is very common in children of that age. He agreed that with iron deficiency, a person is more likely to absorb more lead which may exacerbate the effects of lead exposure.²²⁶ He commented that iron deficiency and overt lead poisoning “go together”; both are associated with age less than five years, city living and lower socio economic status. He also said iron deficiency is more likely to be seen in children with intellectual disability with no lead problems. Further, iron deficiency is very common, whereas lead poisoning is very uncommon.

[148] What complicates the picture is that from about 30 µg/dL and above lead interferes with red cell metabolism and can produce an anaemia which has some features in common with iron deficiency. Anaemia due to lead alone is associated with levels above 60 µg/dL. He said it is also possible that iron deficiency increases lead absorption. He concluded that Manoli’s

²²⁴ T 495.

²²⁵ T 619.

²²⁶ T 620.

anaemia was caused by dietary iron deficiency as it was associated with a low ferritin level. What is less certain is whether the iron deficiency could have produced more lead absorption. To implicate lead in Manoli's iron deficiency, he concluded, is no more than tenuous speculation.

[149] Dr Marraffa told the court that iron deficiency can affect the child's capacity and behaviour such as irritability and lethargy.²²⁷ Further, treatment with iron causes constipation. She was also of the view that the type of anaemia Manoli had was indicative of iron deficiency, not a lead caused problem. She also pointed to the fact that his siblings had iron deficiency and therefore poor diet was the most reasonable explanation. She also said the symptoms of constipation and iron deficiency are more common in the population of children with intellectual disability and autism.

[150] The evidence leads to the conclusion that Manoli's anaemia was based in dietary issues, not caused by lead exposure. I prefer and rely on the opinions of Dr Kilham and Dr Marraffa. On balance, the anaemia was caused by diet.

Observations by medical witnesses about Manoli's physical health

[151] In assessing whether the diarrhoea and other physical symptoms were caused by lead, Dr Duff relied on a history of onset at around three months after moving into the premises.²²⁸ He considered exposure to lead may have contributed to the diarrhoea (at times described by Mrs Koufos as green

²²⁷ T 808.

²²⁸ T 458 – 459.

coloured diarrhoea), alternating with constipation. The history he was given was misleading. Professor Winder said Manoli's health effects indicated long term exposure to lead – of about six months.²²⁹ He too assumed that the symptoms were not pre-existing the move to the premises. He did not look for other causes. He agreed some of the symptoms may have nothing to do with lead.²³⁰

[152] Dr Kilham said the history he was given was not one of gradual onset of toxicity from lead.²³¹ He agreed that if Manoli was exposed to enough lead, he might have started to develop symptoms over six months. He said the symptoms were all extremely common and very non-specific. The fact that Manoli was exposed to lead does not help to conclude the cause. He said there are a thousand diseases which could be characterised by headaches, colic, vomiting and minor regression in behaviour, but the constellation of Manoli's symptoms were not consistent with lead intoxication.²³²

[153] Dr Marraffa did not consider the physical symptoms were classic lead poisoning “at all”.²³³ She said the symptoms were consistent with a wide variety of conditions in childhood; they are possible at blood lead levels of 40 – 80 µg/dL, but unlikely.²³⁴

²²⁹ T489.

²³⁰ T 507.

²³¹ T 587.

²³² T 619 – 620.

²³³ T 841.

²³⁴ T 841.

[154] I appreciate that any changes in behaviour and development must also be considered, however, in terms of the likelihood of lead causing the physical symptoms suffered by Manoli, the medical evidence, on balance, does not support the conclusion that lead was the cause. These symptoms are more likely to have shared the same or similar causes for the ordinary childhood conditions he suffered from prior to moving into the premises.

Observations by witnesses about Manoli's behaviour and development

[155] Mrs Koufos' evidence was that prior to moving into Witherden Street Manoli was a happy child. He was slow with his speech. He would say a few words but she did not notice anything wrong with his motor coordination or any physical problems. He would play with all the toys. He would socially interact with Mrs Koufos when the other children were at school. He would dance to music. When his father came home he would play outdoors, running, kicking a ball and playing with the other children. She did not see any problems with his physical health. All that she noticed was his slow speech. If he wanted a drink of water he would say, (as they generally said at home), "bu-bru" and he would be given a drink. Both the plaintiff and the other children spoke Greek and English at home and hence used "bu-bru" for water and "be-be" for food. She said she knew when he was hungry. She said he was toilet trained at the age of two so there was nothing she noticed that was abnormal. There were no conflicts in the household, (as noted earlier in these reasons, sadly, that was not always the case). He would play with different toys; puzzles, puppets, singing toys,

balls, trains and cars. He would go to her so she could play with him. If she was busy he would run off and get another toy and throw it at her. She could not see anything abnormal.

[156] In very general terms the lay witnesses supported the plaintiff's case suggesting that prior to the move to Witherden Street, speech delay was the only sign of developmental delay; that he played with a wide range of toys and his communication was appropriate.²³⁵ Broadly I accept that the lay witnesses have tried to recall their observations of Manoli but much of their evidence amounts to impressions of a most general kind. In a number of instances they ignored or else were unaware of the factors that may be relevant to the assessment of Manoli's well-being. That is not a point of criticism. The evidence of the paediatricians called indicates it will often be the case that people close to a child will not notice signs of developmental delay especially when the child is very young. Given the lapse of time, lack of details and given the plaintiff is someone with whom a number of them share an emotional association, their evidence is somewhat unreliable.

[157] The lay evidence given was that towards the end of 2005 Manoli began to behave strangely, lost the speech that he had acquired, began to run into walls, ceased to be toilet trained, socialised less and generally regressed. This must be seen in the context of a number of the witnesses saying they

²³⁵ Mrs Koufos T 21-22; Panormitis Koufos T 270-272; Charlie Mailis T 370; Micheal Mailis T 395; Lynette Hoffman T 13-14.

started to observe him more closely after being told he had brain damage.

Dr Ramdoss also observed the change in his behaviour and was concerned.²³⁶

[158] The plaintiff's case is that this reported regression and symptoms were caused by lead. The evidence about the onset of behaviours of concern is not entirely clear. Some of the references given in the lay evidence indicate behaviours consistent with autism prior to 2005. Dr Marraffa's evidence was that it is not uncommon for parents to miss the signs or deny the signs of autism. Once again, parts of histories given to the doctors are either not reliable or have been retracted by Mrs Koufos.

[159] Mrs Koufos gave evidence, for example that Manoli would play with a variety of toys but was not fascinated and so possessed with any one toy.²³⁷ Later she denied this was her evidence, but then appeared to accept this was her experience of Manoli.²³⁸ She disagreed she told Dr Marraffa that she noticed at 12 months that Manoli had no real interest in toys and would just grab toys in a random fashion.²³⁹ Dr Marraffa's history records she said this.²⁴⁰ In Mrs Koufos' evidence in chief she referred to Manoli recognising colours. She would name the colour and he would name the colour.²⁴¹ Later she retracted this saying that he would not say (for example), "blue" straight

²³⁶ Mrs Koufos T 31-35; Mr Koufos T 277; Charlie Mailis T 371-372; Michael Mailis T 396-397; Olga Fountis T 363-365; Dr Ramdoss T 482.

²³⁷ T 21, 22, 55.

²³⁸ T 204.

²³⁹ T 55-56.

²⁴⁰ Exhibit D 30, at 11.

²⁴¹ T 56.

after her. After he played with blocks, for example he would name a colour (blue). She said he never repeated back what she said.²⁴²

[160] Mrs Koufos disagreed she told Dr Marraffa Manoli was really only interested in food and that he would focus on that rather than playing with toys or other children.²⁴³ This denial is contrary to the record Dr Marraffa made. Mrs Koufos agreed she told Dr Marraffa that at six months Manoli would wave to her which Dr Marraffa noted was much earlier than usual. Mrs Koufos did not recall saying to Dr Marraffa that Manoli would use gestures and point to things and drag her to things. Clearly Mrs Koufos described behaviours that were noted by Dr Marraffa that she did not confirm in evidence. The cluster of behaviours described by Mrs Koufos, in part, informed the diagnosis made by Dr Marraffa, contrary to the plaintiff's case.

[161] Mrs Koufos agreed she told Dr Duff that before the age of three Manoli would physically use her hand to communicate or drag her by the clothes. In evidence she said what she meant by this was that if his toys were high he would point to them if he was unable to reach them.²⁴⁴

²⁴² T 203.5.

²⁴³ T 56; 205, 206.

²⁴⁴ T 201-202.

[162] Broadly, other lay witnesses agreed that prior to the move to Witherden Street, this type of pointing was Manoli's mode of communication with adults if he wanted something.²⁴⁵

[163] Mr Koufos agreed that prior to moving into the premises Manoli had delayed speech;²⁴⁶ He was not troubled by this. He agreed that if Manoli did not know what to say he would pull his mother or himself by their clothes or by their hand if he wanted something.²⁴⁷ When asked if he had a choice, would he watch television rather than play with others, Mr Koufos said "he would do both, but he preferred watching Bob the Builder".²⁴⁸ He also agreed that when Mrs Koufos told him that he had brain damage he paid much more attention to Manoli than previously.

[164] Charlie Mailis' observations in relation to changes in Manoli's behaviour are general. He did not notice Manoli's speech delay prior to moving into the premises, admitted to his own memory difficulties "bad memory loss"²⁴⁹ and visited only for brief periods every two to three weeks.²⁵⁰ Similarly, Michael Mailis' observations were of a general nature and the reported changes he noted in Manoli were not observed until after he was told that Manoli had brain damage.²⁵¹ Ms Fountis who, for reasons discussed already, I do not find to be reliable, said Manoli's speech as a young child was not

²⁴⁵ Mr Koufos T 287; Olga Fountis T 365; Charlie Mailis T 376-377; Michael Mailis T 402.

²⁴⁶ T 271.

²⁴⁷ T 287.

²⁴⁸ T 288.

²⁴⁹ T 373.

²⁵⁰ T 369.

²⁵¹ T 398 – 400.

very good;²⁵² she denied he had major problems with his speech before moving to the premises.²⁵³

[165] Lynette Hoffman, a neighbour in the previous home at Groote Street, recalled Manoli was a normal toddler, interacting, playing with toys, not playing by himself.²⁵⁴ Ms Hoffman said that if she said “hello” to Manoli, he would say “hello” back. She did not recall him saying other words, apart from “hello” and “hi”.²⁵⁵ She had not seen him for seven years at the time of the trial. She agreed she may have confused him with another child when speaking of him walking to primary school, which she was told, he did not attend.²⁵⁶

[166] Prior to moving to the premises there are indications of the types of behaviours relevant to a consideration of autism; speech delay; placing or throwing toys at the adult’s feet; pulling an adult by the hand or clothes to communicate needs; echolalic speech. I cannot agree with the submission that aside from speech delay, the plaintiff had no other attributes common with autism prior to his exposure to lead.

²⁵² T 358.

²⁵³ T 358.

²⁵⁴ T 563.

²⁵⁵ T 566.

²⁵⁶ T 565.

[167] Dr Ramdoss, who had treated Manoli for much of his life did observe bizarre behaviour in 2006 that he was so concerned about, he referred Manoli to a paediatric psychiatrist urgently.²⁵⁷

[168] Dr Ramdoss' observations do not mean that Manoli did not exhibit behaviour consistent with autism or developmental delay prior to that time. Although of a different quality to that observed by Dr Ramdoss, the Royal Darwin Hospital notes indicate challenging behaviours on the part of Manoli when he was unwell, being too distressed to be examined, and on one occasion taken home without examination.²⁵⁸ In my view the position in relation to any change in behaviour after moving into the premises is not as clear as suggested by the lay witnesses. Nor can it be readily drawn from the observations of Dr Ramdoss. Dr Kilham's description of how behavioural change would be gradual does not support an inference that what Dr Ramdoss observed was caused by lead. Dr Ramdoss did not wish to be drawn on the question of the involvement of lead. Dr Marraffa's opinion was that behaviours tending to show autism will not always be noticed by GP's who tend to particularly deal with the problem at hand.

Discussion of the diagnosis of autism and an intellectual disability - whether lead is implicated as a cause

[169] Dr Marraffa's opinion is that Manoli has autism and an intellectual disability of unknown aetiology. Although acknowledging that in very rare

²⁵⁷ T 482 – 488.

²⁵⁸ Exhibit 36, Agreed Tender Book, 455-456, 2 October 2003; 12 August 2004 at 445 – 448; 444, 13 June 2005.

cases lead exposure may be a cause of autism and intellectual disability, it is Dr Marraffa's opinion that autism and the intellectual disability suffered by Manoli were not caused by exposure to lead. It is her opinion that the features of autism and intellectual disability suffered by Manoli were present prior to June 2005 when the family occupied the premises. I was impressed with the care, professionalism and honesty of Dr Marraffa's history taking, her reports and her evidence. When challenged, she answered in considered and thoughtful ways; she gave comprehensive answers showing a great depth of knowledge in the field. In my opinion her expertise to make the diagnosis based on the history given, her examination of Manoli and the other material she identified is superior to that of the witnesses called by the plaintiff. Similarly, Dr Kilham is clearly a leader in the field of paediatrics and lead. Other experts seek his opinion in these difficult cases.²⁵⁹

[170] The evidence of Dr Marraffa and Dr Kilham considered with the other evidence in this case leads me to the firm conclusion that Manoli suffered these conditions before he moved into the premises. Dr Samuel Menahem, not called in these proceedings supports their opinions.²⁶⁰ In my view the plaintiff's case fails on the issue of causation. The defendant has well shown that autism and intellectual disability pre-existed exposure to lead. I will discuss the main medical evidence called or relied on in the proceedings.

²⁵⁹ Eg, Dr Edwards, one of the plaintiffs treating paediatricians.
²⁶⁰ Exhibit D31.

Dr Schempp

[171] I acknowledge Dr Elke Schempp took blood tests after Manoli's presentation and one of her conclusions was "mild lead intoxication", however, this does not show, as submitted on behalf of the plaintiff, that she believed lead intoxication played a role in the plaintiff's conditions. Her letter of 4 October 2006,²⁶¹ shows she reviewed Manoli in August 2006 regarding severe speech development delay. She states "The main concern is Emmanuel's speech development. At the age of 4 years he is only using a few single words and mainly using gestures to express himself. He also presents with borderline features for Autistic Spectrum Disorder. His diet is also of great concern. He drinks excessive amounts of milk". On the basis of that short history she has "Performed blood investigations such as full blood count" It is clear she has done a full blood screen, including, but by no means exclusively, lead. Mrs Koufos told Dr Schempp she observed Manoli eating paint. Dr Schempp stated (when replying to Dr Ramdoss and arranging follow up for Dr Edwards),²⁶² the situation was most likely due to an unbalanced diet and that she has given advice to Mrs Koufos. It would appear it was diet that Dr Schempp was focussed on at that time.

[172] On behalf of the plaintiff it was submitted the only form of developmental delay Manoli exhibited before moving to the premises was speech delay that was not uncommon in the Koufos/Mailis family. The speech delay was significant in the case of Manoli, however there were other aspects of his

²⁶¹ Exhibit 36 Agreed Tender Book, 705.

²⁶² Exhibit 36, Agreed Tender Book, 703.

behaviour of concern that were acknowledged by both the lay witnesses and in the history given by Mrs Koufos to Dr Marraffa. Along with the speech delay, there was lack of interest in toys or playing with other children, echolalic speech, gesturing and pulling his mother or other adult and the behaviours observed at the emergency department of Royal Darwin Hospital.

Dr Duff

[173] I have discussed aspects of Dr Duff's reports and evidence already. His research and teaching involves clinical nutrition as it relates to the biochemistry of the brain and the formation of neurotransmitters within the neurons in the brain. Part of that study is directed to chemicals or elements that may interfere with the neurotransmitters. He is interested in lead and other minerals and how they impact at the molecular level on the brain. His clinic specialises in childhood neuro developmental disorders. It involves treatment and testing and he looks primarily at genetics and nutritional factors that affect function.

[174] I have mentioned previously that part of the history taken by Dr Duff does not accord with other evidence. He proceeded on the basis that Manoli was developing normally except for some speech delay. He considered his socialisation was normal and his imaginative play or his interactive play was normal. The history he obtained indicated he was using toys appropriately. Dr Duff recorded Manoli was not suffering from autism prior to moving to the premises. He said speech delay was not sufficient to diagnose autism. He said the stereotypical behaviours of autism were not present prior to

moving into the house. He commented that being in a bi-lingual household may have a bearing on speech delay but generally language “catches up”.²⁶³ Dr Duff was firmly of the view lead caused the conditions Manoli has; and that low levels of lead exposure were associated with developmental deficits.

[175] Dr Duff relied on the American body, the Centre for Disease Control and Prevention (CDC) providing a blood lead level of 10 µg/dL should prompt public health actions, (he points out this is now being revised to recommend 5µg/dL);²⁶⁴ however, it is common ground that 10 µg/dL should not be interpreted as a threshold for toxicity, since no safe blood lead level threshold had been identified. This is similar to the position of the (Australian), National Health and Medical Research Council.²⁶⁵ Further, Dr Duff stated that since 1991, low level exposure to lead during early childhood was shown to be inversely associated with neuropsychological and development deficits in the first seven years of life; he said the threshold for clinical concern of lead exposure in this age group was recently recommended to be 5 µg/dL rather than 10µg/dL.

[176] He suggested that symptoms of lead toxicity begin in children at low lead levels and explained his understanding of how heavy metals affect neurotransmitters in the brain. Dr Duff gave evidence of what he considered to be regressions, including the lack of interaction with others, and being

²⁶³ T 431 – 432.

²⁶⁴ Exhibit D14 at 3.

²⁶⁵ Exhibit D14.

more withdrawn compared with Manoli's previous reported interactivity. In Dr Duff's view, there was nothing else to explain Manoli's symptoms other than lead exposure.²⁶⁶ His view was that if Manoli had continued to play in the area where the testing revealed lead levels of concern, it would have had an on-going affect on his functioning and would have contributed to his disabilities.²⁶⁷ The assumption that Manoli continued to have ongoing access to the suspect area of the yard, unbeknown to Dr Duff, is contrary to the findings here.

[177] Dr Duff excluded autism or symptoms of autism as being present prior to the middle of 2005.²⁶⁸ He also noted the report from the Carpentaria Disability Services indicating Manoli was interacting with others and had imaginative pretend play at that time. I note that while there appear to be occasional positive observations relevant to Manoli's development in the Carpentaria Disability Services report of 22 June 2007, the balance of the observations and assessment is indicative of developmental delay.²⁶⁹

[178] Dr Duff commented Manoli had deteriorated between 2007 and 2008, suggesting this was not typical of what would be expected of a child with autism. His opinion was that an autistic child would usually gain some

²⁶⁶ T 436.

²⁶⁷ T 439.

²⁶⁸ T 442.

²⁶⁹ Exhibit 36, Agreed Tender Book, 323-330.

skills. He said the symptoms were caused by damage to the brain which could be on-going.²⁷⁰

[179] Of this assumed regression, he understood Manoli was toilet trained by two and a half but by 2008 he was no longer toilet trained; he relied on the changes in his interactions, less spontaneity, no reactive play and being more withdrawn. At that time (2008), Dr Duff says he met the criteria for autism.²⁷¹ He said there was an event in his life that caused or appeared to be causing a regression and he believed that to be caused by exposure to lead.

[180] He noted Mrs Koufos had difficulties with her previous husband but they were resolved. She had stressors in relation to looking after the children but he considered Mrs Koufos a resilient, tough lady and he did not think the stress would have any effect on Manoli. No symptoms were consistent with parental stress.²⁷² He thought headaches and regression in cognitive skills also appeared to be consistent with lead. In this regard it would appear there is very little in Manoli's medical history discussed above indicating complaints about headaches.

[181] Dr Duff considered the experience of Dr Kilham to be based at a time when lead exposure was very common in the community and at a time when the levels that were thought to be safe some 30 years ago were no longer

²⁷⁰ T 468.

²⁷¹ T 433.

²⁷² T 434.

considered safe levels.²⁷³ He agreed the symptoms and history suffered by Manoli could not be explained by the highest measured blood lead level of 16.7 µg/dL. Of the extent of the cognitive deficits or the decline in his abilities, Dr Duff suggested that Manoli would have had to have been exposed to higher levels of lead prior to 16.7.²⁷⁴ His evidence was that he had treated hundreds of children with autism and believed in this case lead was responsible for the autism.²⁷⁵

[182] Dr Duff was taken to the history he recorded about altered behaviour three months after moving into the house. It was suggested to him that if the history showed there had been unusual behaviour such as screaming, refusing to do as one's told, urinating without apparent control before as well as after moving to the premises, that there may not be a connection with lead. Dr Duff confirmed his view that on the basis of the history provided Manoli had good socialisation and with his spoken language difficulties, he tended to compensate by pointing. He said this was a compensating strategy that Manoli was developing, but before the move there were no autistic features.²⁷⁶

[183] Dr Duff agreed that speech delay and pulling the hand or clothes rather than eye contact or speech in order to get what a child wants is a factor that would be taken into account in arriving at a diagnosis of autism. He agreed other factors would be echolalia, and throwing toys at the feet of the parent

²⁷³ T 435.

²⁷⁴ T 458.

²⁷⁵ T 443.

²⁷⁶ T 442.

rather than using eye contact. He agreed those factors might also indicate developmental delay and agreed the challenging behaviours described in medical examinations would also be a factor indicating developmental problems.²⁷⁷ He said that in 2008 Manoli met the criteria for autism but in 2007 there were no reported autistic features aside from language delays.²⁷⁸ He said that by 2008 the degenerative process that took place showed he was on the autistic spectrum. He diagnosed “childhood disintegrative disorder”. He agreed the test he used to suggest autism says nothing of the cause nor when the problem manifests.²⁷⁹

[184] In relation to whether there was regression Dr Duff said there may be times when an autistic or developmentally challenged child lacks motivation, so care must be taken when comparing a child who is misbehaving on a particular day as distinct from displaying a genuine capacity. He said he has never seen a child with autism degenerating the way Manoli has. He said damage done to the brain leading to regression between 2007 and 2008 was done through lead as it continues to cause problems because it is stored in the brain and other tissues and is gradually leached into the blood.²⁸⁰ In relation to whether there was a failure or denial to notice the features of autism, Dr Duff agreed it was common that problems with autism are not detected until the child commences schooling.²⁸¹ He agreed it may depend on the capacity of the parent to detect problems. He thought, however, that

²⁷⁷ T 461.
²⁷⁸ T 432, 442.
²⁷⁹ T 460.
²⁸⁰ T 467.
²⁸¹ T 461.

Mrs Koufos had appropriate benchmarks set by her other children to assess Manoli by comparison. He agreed that a stressed parent caring for many other children may not be able to make an objective analysis of this.²⁸²

[185] Dr Kilham's opinion by way of contrast was that the only way for Manoli to have regressions of behaviour would have been if his blood lead level was about 60 to 90 at which level, he said he would have exhibited other evidence of lead poisoning. Dr Marraffa considered childhood disintegrative disorder a "nonsense" diagnosis. She said she has never made that diagnosis; that it is a diagnosis created by a group of psychiatrists who wanted criteria to describe various situations. Dr Kilham agreed with her opinion. Dr Marraffa said childhood disintegrative disorder is not used by paediatricians and she suspects that children so diagnosed, if they were looked at carefully probably had either autism or a neuro degenerative condition not yet diagnosed.

[186] In relation to Dr Duff's diagnosis of childhood disintegrative disorder, Dr Marraffa notes that in the DSM V, (to replace DSM IV), the diagnosis of childhood disintegrative disorder was subsumed into the category "autism spectrum disorder". Childhood disintegrative disorder will no longer be a separate condition. She said even though assumed regression may be coincident with lead exposure, it does not mean it was caused by lead. She said it was possible that regression was consequent on lead poisoning but

²⁸² T 461.

that it was very unlikely. It was much more likely to be due to a different cause.

[187] Although Dr Duff has a clinical practice, he agreed that apart from Manoli he had treated only two children in the past five years who have had exposure to lead.²⁸³ That treatment involved nutritional supplements and neuro-therapy.²⁸⁴ Although Dr Duff has reviewed some of the literature in relation to the effect of toxins on the developing brains of children, he has not written or published in relation to lead. He said he was not a toxicologist but he knew the effects of lead on the brain as he is a neuroscientist.

[188] Dr Duff was asked whether a passage in his first report was taken from Wikipedia. Dr Duff disagreed saying the scientific source of his work was Pearce's work "Burton's Line in Lead Poisoning".²⁸⁵ Dr Duff said that Wikipedia often uses material in sources already published. A passage on page 4 of Dr Duff's first report is almost identical to an extract in Wikipedia.²⁸⁶ The extract in Dr Duff's report is much closer in form to the Wikipedia extract than to the Pearce article. I am not prepared to conclude, as suggested on behalf of the defendant, that Dr Duff lied about this. It is difficult to be confident about the accuracy of his opinions, however, given the disparities in the source material used. In listing the symptoms of chronic lead poisoning Dr Duff omitted a number of symptoms listed such as

²⁸³ T 447.

²⁸⁴ T 106.

²⁸⁵ T 109.

²⁸⁶ Exhibit D15.

numbness and tingling in the extremities; fatigue, problems with sleep, headaches, stupor, slurred speech and anaemia. Dr Duff said he omitted those as they were not relevant to Manoli. Dr Duff completed the relevant paragraph in his report stating, “Note that Manoli suffered from most of these symptoms”. In my opinion his approach has been skewed from the outset in a large part because of the history he was given. Further, the types of treatment regimes he is involved in are not accepted by paediatricians of the calibre of Dr Kilham and Dr Marraffa. I prefer the evidence of Dr Kilham and Dr Marraffa.

Dr Ramdoss

[189] As a result of Dr Ramdoss’ observations of Manoli in 2006, and noting his behaviour was bizarre, Dr Ramdoss referred him to a paediatric psychiatrist. On that occasion Dr Ramdoss spent more than half an hour with Manoli.²⁸⁷ Dr Ramdoss was aware of a speech development problem at around the age of two years, but had seen Manoli and his mother speak in Greek and had seen Manoli responding to her.²⁸⁸ He did not express any opinion on whether Manoli’s problems were related to lead. He understood that the paediatrician, Dr Edward’s view was there was no evidence of blood lead levels high enough to implicate lead.²⁸⁹ He had not observed or been given information about the other siblings having language problems.²⁹⁰ He said he would generally be involved with the children that he saw in his practice.

²⁸⁷ T 484, also discussed at paras [167] – [168].

²⁸⁸ T 491.

²⁸⁹ T 494.

²⁹⁰ T 496.

He would play with them and bring any problems about interacting with the child to the attention of their mother.²⁹¹

[190] In assessing what can be drawn from Dr Ramdoss' report and evidence, I bear in mind Dr Marraffa stated that the fact Manoli's GP did not notice anything until June 2006, (until he displayed the unusual behaviours), was not unexpected. She said GP's and paediatricians are notoriously poor at picking up developmental problems. She said both staff at the emergency department of the hospital and GP's focus on the acute presenting problem and appropriate treatment. They do not make developmental assessments.²⁹² Dr Kilham agreed with these observations.

Professor Winder

[191] I have discussed Professor Winder's evidence about previous blood lead levels above. His view was that ongoing exposure to lead after August 2006 contributed to Manoli's ongoing deficits and neurological behaviours. He agreed with the proposition however that it is simply not possible to accurately identify which ongoing symptoms were caused by the first period, prior to August 2006 and which ongoing symptoms were caused by the second period. He appeared to accept that distinguishing between the two was really a matter for speculation.

²⁹¹ T 497.

²⁹² T 805.

[192] Professor Winder gave evidence about studies that revealed a connection between ingestion of lead and autism.²⁹³ He said lead has been a non-specific inhibitor of brain function and specific diseases like autism may be precipitated by impairment of brain function caused by lead. He acknowledged, however, that two papers he referred to in support of his opinion included children who had already been diagnosed as autistic before exposure to lead.²⁹⁴

[193] Professor Winder said that at a blood lead level of 16.7 µg/dL he did not expect to see the frank signs of lead poisoning, but did expect to see signs of intoxication.²⁹⁵ He thought Dr Kilham's reports about signs of lead poisoning and intoxication were old fashioned and out of date.²⁹⁶ In re-examination he indicated that continuing exposure beyond August 2006 would contribute to Manoli's ongoing neurological deficits and neuro behaviour. Professor Winder, as a toxicologist agreed that he had no expertise in autism and agreed he was unable to comment on the symptoms of autism but did so as there are psychological symptoms common in lead intoxicated children including autism and behavioural problems.²⁹⁷

[194] Professor Winder had proceeded on the basis of a history which must be regarded as flawed, of assuming all of Manoli's symptoms were not pre-

²⁹³ T 514.

²⁹⁴ T 497.

²⁹⁵ T 527.

²⁹⁶ T 527.

²⁹⁷ T 509.

existing.²⁹⁸ Professor Winder was attempting to show an association with lead and did not look for other causes. He agreed a number of Manoli's symptoms may have nothing to do with lead, however, he said that learning difficulties are associated with lead. Tantrums, he said may be due to lead induced behaviour problems.²⁹⁹ Professor Winder agreed that he did not consider the question of pica in Manoli's case in terms of ingestion of lead, but noted he was eating paint flakes from the house.³⁰⁰ He agreed that if there was no ongoing exposure to elevate the blood lead level, and the blood lead levels fell, there may still have been lead in the body having an effect and causing symptoms between October 2006 and May 2007, but those symptoms could only be the consequence of exposure to lead in the first period.³⁰¹

Dr Knight

[195] Dr Knight was unable to attend the hearing to give evidence through illness. His reports were received into evidence.³⁰² Dr Knight has an interest in research in relation to blood lead levels. His opinion was that a low blood lead level, even below 10 µg/dL, could cause brain injury. He expressed a view that the deterioration in Manoli was consistent with exposure to lead. He too, adopted "Childs Disintegrative Disorder" as a developmental disorder that should be considered. Although admitting his report, on such a contentious issue and bearing in mind the context of other evidence in the

²⁹⁸ T 499.

²⁹⁹ T 506-508.

³⁰⁰ T 515.

³⁰¹ T 511.

³⁰² Exhibits P18, P19.

case, I cannot give his evidence weight of any significance. The inability to cross-examine leads to significant disadvantage. His was the only medical opinion before the court supportive of the plaintiff's case. The highest his opinion is put, however, was to suggest Manoli's condition was "coincident with his exposure to lead". I do not find Dr Knight's opinions persuasive. I do not ignore that Dr Knight reported to Dr Kilham as head of the Division of Medicine at Westmead Hospital.

[196] The medical case on behalf of the plaintiff is not strong.

Dr Kilham

[197] Dr Kilham saw the plaintiff on 9 June 2007 having been referred from Dr Edwards. Most of the children Dr Kilham treated, were from 1967 onwards and related to lead paint in old housing. Those children had very high lead levels of 70 – 130 µg/dL. Dr Kilham recalled some deaths of children he treated. He was involved in a working party of the National Health and Medical Research Council 30 years ago that played a major role in public decisions to gradually remove lead from petrol. In a 30 year period he had seen many children with blood levels indicative of "abnormal lead exposure" but most of them had much higher levels than Manoli's highest blood lead levels. In most instances the children had no signs or symptoms of lead toxicity.

[198] He thought it reasonable to state that there was some lead contamination of the Koufos family's premises and that Manoli had one blood lead level

which was just high enough to suggest some undue lead exposure. He also said it is apparent that Manoli is autistic. He said anything beyond that conclusion is subject to argument and speculation. He relied on the developmental assessment conducted by Dr Marraffa who concluded “Manoli suffers from autism with an intellectual disability”. From the history he concluded that there were clear cut indications of autism prior to the move to Nakara in June 2005. He also thought it common for parents to be seemingly unaware of even quite significant developmental delay in young children. He said he is also aware that lead poisoning may be a rare cause of autism but that it was irrelevant in Manoli’s case given the recorded lead levels and particularly if evidence of autism pre-dated the excess lead exposure.

[199] He noted the conflicting histories and that Manoli’s mother had been certain of lead poisoning ever since the level of 16.7 µg/dL was revealed. From the various histories he records that he was told Manoli was playing and “covered in ash” at times. He was told Manoli was seen eating paint flakes that were later shown to be lead free. He said lead intake would have been via Manoli licking dust from lips and hands, and leaded dust on food or other objects put in his mouth, common to the pica disorder. He said lead intake would be a slow process, as even with heavy lead contaminated soil it would have taken many weeks or months to get to around 60 – 70 µg/dL at which point Manoli would have developed constipation, abdominal pain, and subtle behaviour changes. The symptoms would have an insidious onset,

gradually progressing only if leaded dust was constantly available.

Diarrhoea would be unusual. Constant irritability or severe apathy would have suggested progression to lead encephalopathy, but as he noted, the history suggested that this did not occur. He thought the illnesses reported were not what would be expected with lead poisoning.

[200] He said it can be stated with certainty that a blood lead level of 16.7 µg/dL, even if sustained for months or years would not have produced the illness described.

[201] He points out that while Dr Duff states that damage from lead poisoning would be untreatable, Dr Duff considered that some improvement may have been due to “experimental nutrition therapy”. Dr Kilham expressed the view that these are unproven therapies.

[202] Dr Kilham explained the concept of the target level (10 µg/dL) for Australia is widely misunderstood as a “safe” level and as a level above which “lead intoxication” is present. He said it is referred to as a “target level” as a maximum for all Australians and is based on the thinking that it should be “way below” levels which cause lead poisoning.

[203] He referred to subsequent epidemiological studies showing an association between lead and neuro-cognitive effects. This does not in his view imply a causative effect but is an indication to aim for the lowest possible lead levels across the whole population. As the levels are most useful for public policy reasons, he agrees that activists play an important role in bringing

attention to lead risks. He sees it as unfortunate that many now refer to any lead level over 10 µg/dL as “lead poisoning”. He considers this a distortion of the word “poisoning”, that while well intended, it does harm to families where levels above 10 µg/dL are discovered. He considers this is well illustrated in Manoli’s family.

[204] He considers the NHMRC information paper on lead to give a fair and balanced concept of a target blood level of 10 µg/dL. He says there is no doubt about what level of blood lead equates to overt effects in individuals. Laboratory changes appear from around 40 µg/dL and mild non-specific and subtle changes from around 60 µg/dL. Emotional and behavioural symptoms begin to appear at these levels. Dangerous lead poisoning is associated with levels over 100 µg/dL with seizures and coma; death is common. With aggressive therapy children may recover completely, and have no brain damage. Intellectual disability can also result from persistent sustained high blood lead levels, short of the levels associated with lead encephalopathy. He acknowledged this is what he understood is being claimed in regard to Manoli, although he expressed an opinion that this is based on speculation as opposed to sound evidence.

[205] In relation to very low levels (<5 µg/dL) and ascending amounts, he said the variety of epidemiological studies from about 30 years ago show an association between lead levels and neuro-cognitive deficits which are greater as lead levels rise, but which are not linear. The studies in support

attempt to balance out confounding factors but a definite causative link between lead and neuro-cognitive deficits has not yet been established.

[206] Dr Kilham also considered bone X-Ray changes that may implicate lead were considered in Manoli's case as no more than weak, inconclusive evidence of lead poisoning. He concluded that Manoli's condition is not caused by exposure to lead.

[207] Dr Kilham made a number of concessions during the course of his evidence, for example he accepted that while he did not believe lead was a cause of autism, he agreed it might be a major or contributing factor in an extremely small number of children diagnosed with autism.³⁰³ He accepted that very high blood lead levels could produce the pattern of brain injury that caused autistic symptoms.³⁰⁴ He also conceded in terms of the half life theory, that earlier in time, Manoli might have had a much higher blood lead level than the recorded blood lead level. I do not however take this as Dr Kilham resiling from his firm opinion that to attempt to conclude the earlier blood lead levels was speculative. He also agreed there was an association between low blood lead levels and behavioural problems,³⁰⁵ however consistent with the tenor of his opinion, disagreed that this was any more than an association, in the sense that it did not establish causation. He similarly accepted there was a relationship between blood lead levels and

³⁰³ T 645.

³⁰⁴ T 637.

³⁰⁵ T 630.

attention related behaviours supported by multiple studies.³⁰⁶ Dr Kilham also agreed that currently epidemiological studies suggest that lead in much lower levels than was thought previously could be producing damaging effects on young developing brains.³⁰⁷ His point was that this does not allow a conclusion to be drawn in relation to one particular child who suffered apparently a very severe effect on his brain going from being not too bad to being very bad and on some claims is due to lead.³⁰⁸ He agreed this was an evolving area. His point was that the studies now needed are the ones which would more clearly establish causation.³⁰⁹

[208] Questions critical of the way Dr Kilham dealt with the issue of maternal stress were put to him, however all that he was suggesting was that he did not think that the stress or stressors that he recognised, (and he agreed here with Dr Menahem), had anything to do with the question of lead.³¹⁰ He expressed the view that Manoli's family would have been more stressed than an average family but no firm conclusions could be drawn that relate directly or indirectly to the lead poisoning question. He agreed his opinion was partly conditioned upon the findings of Dr Marraffa based on her examinations of Manoli.³¹¹

[209] Dr Kilham was questioned critically in relation to his history taking. He said the details of his consultation with Manoli were fresh in his mind when

³⁰⁶ T 631.

³⁰⁷ T 628.

³⁰⁸ T 628.

³⁰⁹ T 628.

³¹⁰ T 604.

³¹¹ T 626.

he prepared the report. The consultation took about one hour. He set out in the initial letter the history that he took from the mother. He was trying to explore the overall situation. He briefly talked to the mother about Manoli's developments and milestones but it was already clear through a developmental assessment that he was not developmentally normal in 2007.³¹² He commented that Mrs Koufos' understanding that developmental delay might be temporary is wrong because it is not reversible.³¹³ He did not mention maternal stress in that report but he said that does not mean he did not consider it. He thought it obvious and that is why he wrote to Mrs Koufos a few days later.³¹⁴ He understood his job was to consider the lead, not every single symptom in the context but the constellation and overall pattern and then try to reassure Mrs Koufos that the problem probably wasn't due to lead but that she was doing the right things. He found Mrs Koufos a very attentive mother but she had a huge amount on her plate. He rejected that he missed something through being in a hurry. He told the court that if he had received a history which was consistent with medium range lead poisoning he would have said so.³¹⁵ He said that if Dr Marraffa's views were left out, he would have persisted with his view that Manoli had some form of global developmental delay or a type of autism. He said

³¹² T 586-587.

³¹³ T 588.

³¹⁴ T 602-603; Exhibit D 25.

³¹⁵ T 623.

diagnosis of autism can be made later than when the child is aged three years, if the criteria were manifest before that time.³¹⁶

Dr Marraffa

[210] While Dr Marraffa openly acknowledged mistakes could be made when taking a history, she had a significant amount of time during the particular consultation and spent two hours with Mrs Koufos and Manoli; she asked open-ended questions in accordance with what she considered to be sound practice; she took detailed notes, had ample time because she was in Darwin for a conference and could write “the bones” of the report straight away. Where there is a conflict between the evidence given by Mrs Koufos about what she told Dr Marraffa and the history Dr Marraffa says she took, I prefer the evidence of Dr Marraffa.

[211] In terms of Dr Marraffa’s opinion that Manoli has autism and an intellectual disability of unknown etiology, it is clear she took a detailed history and had access to previous reports. She made a number of observations in relation to the unusual aspects of history taken from Mrs Koufos: for example, that Manoli was smiling and looking at her in the first two weeks of life and by one month giggling at her. Dr Marraffa said this was early compared to the expected age. She was told he was waving bye-bye at six months, that also is much earlier than the usual time. He would pull her by the trousers or take her hand or take her to get what he wanted at 18 months and this continued until well after he was three years old. At two and a half he used

³¹⁶ T 648.

certain learned rote phrases such as “what’s this” and that by two years and eight months he could name colours if asked “what colour is this?” Dr Marraffa stated this was not the usual development pattern. Children first usually are able to identify colours when nominated by another and then they are able to name a colour when asked by another “what colour is this?” She concluded Manoli’s language development was definitely delayed before he was three years old.

[212] Mrs Koufos also reported that at 12 months of age he had no real interest in toys and would just grab toys in a random way. He watched the television especially “Bob the Builder” and “Nickelodeon Kids”. Mrs Koufos would play with him with a tip truck and he would shovel dirt into it. Mrs Koufos told her he was really only interested in food, would eat everything and that food was his focus rather than playing with toys or other children. She noted the report from Dr Schempp indicating he ate very little because he drank vast quantities of milk and thus became very iron deficient. Mrs Koufos reported he was toilet trained, with bladder by 18 months and by two years able to pass bowel movements into the toilet. He stopped using a night nappy by 22 months.

[213] Mrs Koufos told her the family moved into the home where he was exposed to lead in June 2005 and her youngest son was born in July 2005. He had vomiting everyday and diarrhoea which she said was fluorescent in colour and this occurred on and off over the next year. She reported he started biting his nails and he had headaches by Christmas 2005. By Greek Easter

in 2006 when an uncle visited, he was distressed with marked tantrums, stripped off his clothes and completely lost any speech he had and became incontinent. He also started to spin wheels on toys and would slam himself against walls; Mrs Koufos said it was at this time that he started to bite the back of his hand.

[214] Dr Marraffa said there was no history of age appropriate social interaction with peers or pretend play at this time and it was reported he only started to demonstrate some imaginative play, aged eight. In August 2006 within the first week of preschool the teacher said that he was aloof and non-communicative and in need of help. At the end of August 2006 a severe iron deficiency was found and the mildly elevated lead level found. He went into transition at the local primary school and due to the school locking him in a room his mother went to the Anti Discrimination Board to get him extra help. He now goes to a special school. He has limited skills in all language areas; receptively, pragmatically and expressively. He still takes people by the hand to get what he needs. He calls his mother “mum” but does not refer to himself as “Manoli” or “I”. He has repetitive and limited play skills. He uses echolalia. At home his favourite and preferred activity is to fill the small pool with water and then splash it.

[215] On examination of Manoli Dr Marraffa noted echolalia. She did not hear many sentences spoken by Manoli throughout the two hours she spent with him. He pushed her hand away firmly when he was stopped from taking a toy from a container. He moved around the room backwards and forwards,

noticing birds on a nearby roof and then watched the blades of a fan spinning for a short time. Dr Marraffa made observations of Manoli completing a construction task. The behaviour exhibited was typical of a child with autism. Typically he removed her hands forcibly to get the pieces that he wanted to use to complete the task. He used avoidant behaviour to get out of doing the task presented. Repetitive play was noted as was acting out some aggressive behaviour between dolls. He could not engage in a two way conversation and the only conversation with his mother was around having his needs met. Reciprocal interactions were not observed, although his mother had a very good ability to relate to him. To get his attention by calling his name failed. This was also so with his mother. His motor skills showed he was clumsy, flat footed and slow. Mrs Koufos told her he rides a bicycle with training wheels. He had difficulties getting up from the floor and could not run fast. He could only go up and down stairs holding onto a banister and had auditory sensory abnormalities. He showed no response to pretend crying. The history, assessment and information made available led to the diagnosis of autism and intellectual disability.

[216] In relation to a cause, Dr Marraffa said the cause was unknown. From the history his mother gave her, the developmental difficulties were present before the family moved to the house and well before the exposure to lead. Mrs Koufos reported to her that Aphrodite had severe language delay up to seven years of age. She reported also that her son who was 18 months younger than Manoli had severe problems when he started school with

behavioural difficulties. Two of her own brothers had severe language delay and some of her nieces and nephews were also reported to have language delay.

[217] In a supplementary report Dr Marraffa describes autism as a set of behaviours that are neuro-developmental in origin; it is a triad of impairments in language, social interactions and behaviour demonstrated by limited interests, obsessions and rituals. It is recognised by the quality and intensity of behaviours observed in a child when compared to typically developing children. Autism is not an intellectual disability although in 50 – 75 percent of people with autism, intellectual disability co-exists. In Manoli autism is distinguished by his poor language skills compared to peers, his lack of social language and his use of repetitive and echolalic speech. He has not established peer relationships and socially he is not able to maintain a conversation. His inability to seek help or initiate interactions are also features of autism. His repetitive behaviours and the need for sameness and lack of creativity are the third part of the triad. He has sensory problems such as licking his fingers, more recently biting the back of his hands and was observed to put his hands over his ears when certain noises were made.

[218] Intellectual disability refers to a difference in a person's rate of learning new skills and involves a comparison with same aged peers. As children get older the gap in skill acquisition gets wider.

[219] Autism is a biologically based neuro-development disorder with diverse and complex causes. In most children currently, no cause is identified. It is likely the interactions between multiple genes cause autism. Lead exposure is a rare cause of autism.

[220] In a further report Dr Marraffa concedes that if Manoli was exposed to high levels of lead in the soil and thus potentially higher levels in his blood then he may have had an added insult to an already vulnerable brain which was not developing typically before the exposure. This is clear in her evidence though she does not accept the anterior method used to calculate a higher lead level utilised by Professor Windler.

[221] Dr Marraffa agrees with the DSM V entry that states that the diagnosis of autism can be made if symptoms are present “in early childhood but may not become fully manifest until social demands exceed limited capacities”.

[222] Dr Marraffa disagreed that Manoli’s symptoms are better explained by lead poisoning. She also disagreed with Dr Duff’s statement that “although there was delay in development of spoken language, Manoli compensated through alternative modes of communication, such as gesture or mime or physically using the hand, clothes or other body part of siblings and adults for communication”. Dr Marraffa made a distinction between children with a simple “delay of spoken language”. She says they use their facial expression, proto-declarative pointing and reciprocal gesture rather than taking an adult by the hand or clothing to get what they want. She

concludes that the communication attempts Dr Duff described in Manoli are highly suggestive of autism. From the history she took she said it was clear Manoli had significant receptive and expressive language and communication impairments. Not pointing or using eye contact but rather pulling the mother's shirt or dragging her by the hand to get what he needed was occurring before he was three and much before lead exposure occurred. That is the difference, she pointed out, with autism, and with "delay of spoken language".

[223] Dr Marraffa agreed she was dependent upon the mother's observations of Manoli but said she was looking at a number of issues to form her opinion. She agreed it was not unusual for people to have language problems which ultimately resolved.³¹⁷ In relation to the question of being a member of a bilingual household she said bilingual children do not have language problems, rather if the child has normal intellect they can learn three languages and there may be a very slight delay in their expressive language but no difficulties in receptive language or the pragmatics of language.³¹⁸ Dr Marraffa left open the possibility that mother's response about colours was not the indication that she took it to be. She agreed that in her judgment the mother was a poor historian³¹⁹ however clearly Dr Marraffa asked questions in a way that would allow her to take an accurate history. That was all part of her professional judgment. She said she had no

³¹⁷ T 823.

³¹⁸ T 824.

³¹⁹ T 859.

difficulty making a diagnosis of autism. She said many parents do not recognise the symptoms of autism until the child is subjected to social demands beyond the child's capacity. This will be reflected in DSM V. The various modes of communication used by Manoli informed her opinion.

[224] When cross-examined to the effect that the view of the mother about Manoli was not as Dr Marraffa reflected in the history, but rather was that apart from language problems there were no other matters of concern, Dr Marraffa explained that it is not necessarily the case that classic autism will be obvious to the mother or health professionals. She disagreed that she could not have come to the diagnosis if any of the elements of her history were incorrect. If he had none of the symptoms or concerns, he would have been developing normally and if the only relevant symptom was language delay, there are many explanations apart from autism which might explain that. She went into further detail to explain that if it was language delay across all three areas of language function then she would seriously need to look at autism, intellectual disability or a specific language disorder. If he had imaginative and creative play that would suggest it was not autism.³²⁰ If he made good eye contact and was not noticeably socially unskilled that would suggest that autism was not a possible diagnosis. Children with autism generally use proto-imperative pointing rather than proto-declarative pointing. Dr Marraffa said if her history does not stack up against the facts,

³²⁰ T 828-829.

then the history was not reliable but she has no doubt Manoli was demonstrating symptoms and signs consistent with autism before age three.

[225] As Dr Marraffa said, it depends not only on the history but her clinical judgment, 30 years of seeing children with parents who are variably skilled at giving the history and making a judgment about what the history means. If Manoli presented with delayed language at three, autism would still need to be considered with other conditions. If at age three in every other respect he was not showing a disability then an alternative would have to be considered.³²¹ Although Dr Marraffa said it was possible that lead was a contributor it was highly unlikely. Even blood lead levels of 40-60 are not enough to explain the degree of disability seen in Manoli. Her view was that in order to suffer this degree of intellectual disability and autism, Manoli would have needed to have present with an encephalopathic picture requiring urgent chelation therapy and hospital.³²² She said her understanding of the literature was that children who presented with autism and intellectual disability with lead as the cause have presented with seizures, or swelling of the brain that required urgent medical attention.

[226] She said she knows from research that if video footage of children whose parents report their behaviour was normal is shown to people knowledgeable about the early symptoms of autism, they will be able to see the symptoms. The early symptoms of autism are very subtle and they can be completely

³²¹ T 840-845.

³²² T 847.

missed. Parents often don't see what's in front of them. When cross-examined about pretend play Dr Marraffa said she saw rudimentary evidence of pretend play that might be expected in a child of 15 to 18 months. She understood the Carpentaria Disability Services reports and reports from the preschool supported her view about what Manoli was like. When an activity was motivating for Manoli at age five, he was demonstrating pretend play but that was with a skilled therapist.³²³

Epidemiological Studies, lead level guidelines and causation

[227] It is common ground that there is no safe blood lead level. Dr Duff said the NHMRC level was currently being revised from 10 µg/dL to 5µg/dL. In the United States, the CDC has a recommendation to lower the intervention level to 5µg/dL. I accept Dr Kilham's evidence that the reduction is more political, (using that term in the positive, as opposed to pejorative), rather than medical as a means of ensuring that more children with minimal exposure are identified and their environments examined before there is further exposure. Broadly I accept his evidence is consistent with the tenor of the NHRMC guidelines already referred to.³²⁴

[228] The relevant epidemiological studies and the medical experts called accept there is an association between low blood lead levels (around 10 and

³²³ T 836, 858,
³²⁴ Exhibit D14.

5µg/dL) and cognitive, developmental and behavioural problems.

Epidemiology uses “association” rather than “causation”.³²⁵

[229] Although I accept that epidemiological evidence may in certain circumstances be capable of establishing causation in an individual case,³²⁶ the epidemiological evidence, in an individual case still falls to be considered with the other available evidence. In my view this is not a case of inconclusive medical evidence to be supplemented by epidemiological evidence. The epidemiological evidence is not of a quality here that could establish causation. It can only be considered with the other evidence. It has not been established that Manoli’s conditions or health began to deteriorate after exposure to lead. Dr Kilham’s opinion which I accept, was that the differences found in the studies across thousands of children between the developmental progress and intelligence and behavioural problems in children based on varying lead levels, do not mean anything in regard to an individual child. The problem with elevating association to causation is that the effect of confounders, which may be numerous, cannot be determined. In any event, unlike studies conducted over a large group of children over five years, it is inappropriate to apply this work to Manoli. Dr Kilham commented further saying it would not be expected to see a child go

³²⁵ Professor Winder, t 485.

³²⁶ *Seltsam Pty Ltd v McGuinness and Anor* [2009] 49 NSWLR 262.

from being normal to near normal to having a very severe intellectual disability over a one year period.³²⁷

[230] Although recent research has found that very low blood lead levels are associated with IQ deficits, learning and behavioural problems, it would be inappropriate, according to Dr Kilham to apply that to Manoli's case. As Dr Marraffa noted, any reduction in IQ of a few points, (as indicated by studies relating to low blood lead levels), are functionally largely irrelevant in Manoli's case given his intellectual disability.³²⁸

Summary of findings and conclusions

[231] I have made a number of findings throughout these reasons. The findings that follow should be read co-extensively with the reasons and findings already given. The findings are made on the ordinary civil onus, on the balance of probabilities. I do not agree this is a case that warrants any enhanced burden of proof by applying *Briginshaw*, contrary to what appears to have been suggested in submissions on behalf of the defendant.

[232] On 22 June 2005 the plaintiff's parents entered into a tenancy agreement with the defendant. The defendant, a public housing authority for the Northern Territory, had in place a reasonable regime for inspecting its premises to be leased to tenants. It had a reasonable regime in place to attend to maintenance including safety issues. This applied to the premises

³²⁷ T 582.

³²⁸ T 819, 843.

here, 35 Witherden Street Nakara. The plaintiff and his family commenced living in the premises from 29 June 2005.

[233] At that time, Territory Housing were not aware of any risk to the health or safety of the occupants. Nor were they aware, through a history of inspections of the premises, of the presence of any lead contaminants. Had Territory Housing been notified of possible contaminants, (such as batteries, rubbish or ash), they would have arranged to clean them up prior to any new tenancy. The defendant was not in breach of any duty to carry out an adequate inspection of the premises prior to them being leased. The defendant did not fail to properly clean the premises of contaminants that would be observed on reasonable inspection. The defendant was not notified by the plaintiff's parents of the presence of rubbish, batteries or other potential contaminants upon them moving into the premises.

[234] The plaintiff suffered various medical conditions before moving into the premises. Although some conditions suffered by him have similarities with symptoms of lead exposure, exposure to lead did not cause those symptoms or conditions.

[235] In as much as a shift in the factual onus is suggested by application of *Watts v Rake*,³²⁹ in my view the probabilities in any event favour the conclusion that the plaintiff's medical condition was not altered or did not deteriorate by the move to the premises or the exposure to lead. If the defendant here

³²⁹ (1960) 108 CLR 158.

bears the onus to prove any condition suffered by the plaintiff was pre-existing, that onus has been discharged. Much of the hearing was directed to this very question.

[236] The physical symptoms suffered after 22 June 2005 were not significantly different to those suffered prior to that time. Further, the plaintiff's behavioural and developmental characteristics consistent with autism and intellectual disability were manifest prior to him and his family moving into the premises. The plaintiff has autism and an intellectual disability. He had these conditions, albeit at that time undiagnosed, prior to moving into the premises.

[237] The defendant was put on notice of a possible lead source in the premises, on or about late September or October 2006, by being advised of the plaintiff's blood lead level of 16.7 µg/dL (.79 µmol/L), above the NHMRC recommendation of 10 µg/dL, that should result in an investigation. This level does not suggest lead poisoning or toxicity but was sufficient to put the defendant on notice that a reasonable response required inspection of the premises for possible lead sources and testing. The house paint, that the plaintiff's mother's told the defendant the plaintiff was eating, was not a source of lead. The inspection was undertaken in a timely manner and was a reasonable response to being informed of a child in the premises having a blood lead level of 16.7 µg/dL. Soil sampling was carried out in two areas, one of which was from the backyard of the premises where the plaintiff's mother indicated the plaintiff played. It was not the same area or if it was,

it was not in the same condition as the area identified by the plaintiff's parents as containing rubbish and batteries when the plaintiff's family moved into the premises. It was a small darkened area not containing ash, but small pieces of charcoal, recent in appearance, not suggestive of lead.

[238] Sufficient information was communicated to the plaintiff's mother to indicate the plaintiff should not play in the area. The plaintiff's mother knew this and kept the plaintiff away from the area. The plaintiff's blood lead levels commenced to fall and continued to fall after this time.

[239] The defendant arranged for the soil samples to be tested for lead by NTEL on 31 October 2006. The report was received by the defendant in March 2007 indicating levels of lead in the backyard of the premises of 449 ppm, above the level of 300 ppm, the accepted public health investigation level. The defendant failed to advise the plaintiff's parents of the results until 15 May 2007. In not taking steps to obtain the results earlier than March 2007 and by delaying providing the results to the plaintiff's parents, the defendant breached its duty of care to ensure the premises were safe after being notified of a potential hazard. This breach, however, did not result in the plaintiff continuing to be exposed to lead contamination after the soil samples were collected in October 2006. The plaintiff's mother was offered alternative accommodation or excavation of the soil in May 2007. She declined to accept that offer and stayed in the premises.

[240] There is no proven correlation between the level of lead in soil and blood lead levels. On balance, the soil in the backyard was the source of the plaintiff's blood lead levels. It has not been proven that the soil lead level has at any time been greater than 449 ppm. It has not been proven that the plaintiff's blood lead levels were at any time greater than .79 $\mu\text{mol/L}$ (16.7 $\mu\text{g/dL}$).

[241] The plaintiff's exposure to lead did not, on the balance of probabilities, cause or contribute to his medical conditions. Those conditions were suffered by him before moving to the premises. The plaintiff's symptoms were not consistent with lead poisoning. He did not suffer from lead poisoning or lead intoxication. The plaintiff's parents and others close to him did not recognise, or were in denial about the more serious of his conditions. They were content to think it was solely 'speech delay' that would resolve in time. Regression has not been established and if there has been regression it was not caused by lead. Since receiving the first report of the plaintiff's blood lead level, the plaintiff's mother has been convinced that lead is the cause of his conditions. Her strongly held belief about this is not correct.

[242] Manoli and his family need and deserve the support and care that the community can reasonably give. Both Dr Marraffa and Dr Kilham consider the care and treatment available in Darwin, particularly from Carpentaria Disability Services to be some of the best in Australia. It is hoped Manoli's family will continue engaging with the services currently available.

Orders

[243] The plaintiff's claim is dismissed.

[244] Judgment is entered for the defendant, the Northern Territory.

[245] The matter will be listed at a convenient time to deal with any cost issues.
