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Submission #622

FURTHER SUBMISSION TO THE SCIENTIFIC INQUIRY INTO HYDRAULIC FRACTURING.

1. Baseline studies

I cannot express enough my disappointment regarding the current recommendations regarding baseline studies.

It was noted in 15.1 Introduction that:
"Without an adequate **pre-disturbance** baseline, the magnitude of any post-development change cannot be effectively predicted or its impacts assessed."

Despite that awareness by the committee there is no recommendation that baseline studies should be done before disturbance.

In fact there is not even a consistent, unambiguous recommendation that baselines are done before a production licence is **granted**.

For example
Recommendation 9.3 That baseline monitoring of methane concentrations be undertaken for at least one year prior to the commencement of shale gas production on a production licence.

There is no recommendation that I could find for many of the health based air toxin baselines to be done at all (BETX NOx particulates VOCs PAHs formaldehyde etc).

With regard to 15.2.5 Public health
Baseline data needs to be obtained on the frequency and duration of the occurrence of symptoms commonly associated with irritant substances (for example, sore eyes, respiratory irritation, asthma).
This obviously needs to be done in an undisturbed environment before any drilling / fracking/ flaring, (on an exploratory licence or otherwise).

Considering all the evidence that has been presented to the committee it is indeed worrying that the fundamental and critical recommendation to have pre-disturbance baselines has not been made.

2. NICNAS

I am aware that the Inquiry along with many concerned Australians, had been holding out for NICNAS's long awaited report on the safety of chemicals used in drilling and fracking.

For the benefit of the panel I attach below, with permission, a personal communication from Dr Mariann Lloyd-Smith from the National Toxics Network in which she summaries the manifest deficiencies in NICNAS's assessment.

“Australia’s assessment of CSG chemicals

Since 2011, NTN has campaigned for comprehensive assessment of hydraulic fracturing chemicals and those used in drilling by the unconventional gas industry. In mid 2012, the Australian government announced the National Assessment of Chemicals Associated with Coal Seam Gas Extraction in Australia would be undertaken. It was released in December 2017.[1]

Surprisingly, the study focused solely on the above-ground (surface) handling of 113 chemicals used for drilling and hydraulic fracturing for coal seam gas in Australia (but only in NSW and Queensland). Chemicals were identified through an industry survey, direct requests for information made to companies involved in the Australian coal seam gas industry and by reviewing the limited publicly available information (NTN’s UG Report of 2012). The report identified 113 chemicals used for coal seam gas extraction in Australia during the period 2010 to 2012. Industry reports that 59 of the 113 chemicals that were being used in coal seam gas extraction in 2010-12 were still being used in 2015-17, which suggests they have been replaced with other products not involved in the assessment.

While the UG industry was deeply involved in the assessment, affected residents, NGOs and the wider civil society had no opportunity for input. Much of the data provided by industry to the assessment is claimed as confidential business information and therefore, secret.

The Assessment was limited to impacts from ‘above-ground (surface) handling’ and did not assess:

- *potential risks from chemicals entering deeper groundwater,*
- *chemicals in the coal seam or rock that are mobilised by the fracturing process*
- *fugitive emissions and ambient air contaminants*
- *shale or tight gas extraction*
- *toxicity of the mixtures of chemicals*
- *potential effects on agriculture or the food chain*
- *site specific risks of chemicals*

The study concluded that if there were no protective measures were in place 40 of the 113 chemicals tested could potentially cause harm to the health of people using contaminated water either from a leak from a storage pond or in the event of a large transports spill. The study dismisses these risks arguing the transport and use of industrial chemicals and the storage of waste water is strictly regulated by State and Commonwealth governments and this protects the community from any harm.

Groundwater modelling

A 'desktop' study "Deeper groundwater hazard screening for chemicals used in coal seam gas extraction"[2] was also released. This study modelled 13 chemicals, supposedly representative but limited to those with reliable data. Only a fraction of Australia's industrial chemicals have been assessed and have 'reliable data.' Importantly, sublethal toxicity effects of contaminants such as endocrine disruption were not considered nor were the effects of mixtures of contaminants as the ecotoxicological data to do that was not available.

The study estimated the concentration of a chemical when it reaches a water-dependent asset such as a water bore or a groundwater-dependent ecosystem. The Predicted environmental concentrations (PECs) for individual chemicals are then used to assess risks to human health and environment. Industry's concerns over PECs was rumored to have held up the study for so long. The study concluded that dilution would reduce the concentration of chemicals reaching places where people and the environment may come into contact to an acceptable level. It dismissed concerns about possible exposure pathways as either unlikely or extremely unlikely to exist despite the industry reporting exactly these sort of incidences e.g., bore contamination as having occurred.

The study was also based on significant 'assumptions' regarding breakdown products and persistency, e.g., the very persistent brominated biocide, Bronopol has been found to degrade to a range of very toxic byproducts (eg formaldehyde, 2-hydroxymethyl-2-nitropropane-1,3-diol (tris), 2-bromo-2-nitroethanol, bromonitromethane, nitromethane (bromo-nitroethane, bromo-ethanol, and bromo-nitroethanol.) For the purpose of this study, it was assumed that bronopol will degrade to formaldehyde only, thereby dismissing the potential risks of these other highly hazardous metabolites.

Endocrine disrupting compounds

The failure of both of these studies to review the impact of endocrine disrupting compounds (EDCs) used by the gas industry is a major omission, particularly as the studies identified EDCs as being used by the Australian UG industry, e.g., ethylene glycol, bronopol, ethylene glycol monobutyl ether.[3] Chemicals associated with unconventional oil and gas (UOG) have been shown to block or antagonise hormone receptors, particularly androgen and estrogen receptors (antiestrogens, antiandrogens).[4] Prenatal exposure to anti-androgenic EDCs like ethylene glycol, can lead to delayed sexual development and birth defects such as hypospadias. Prenatal exposure to ethylene glycol-methyl cellosolve (ethylene glycol monobutyl ether, 2-Methoxyethanol) can lead to reproductive damage, congenital birth defects, intrauterine growth restriction and death, while perinatal exposure to toluene can reduce serum testosterone in rats. Perinatal exposure to EDCs has been

shown to cause permanent changes in the brain and effect behaviour, obesity, fertility, cancer and result in other adverse health outcomes in laboratory animals depending on the timing of exposure. Some impacts may be inherited and passed through epigenetic [5] changes that may not become apparent for many years. [6]

US studies[7] have found surface and groundwater near unconventional gas activity contained EDCs and had moderate to high levels of EDC activity. Samples taken from sites with little drilling showed little EDC activity.

Despite the over five year wait, the industry continues to use unassessed hazardous substances for fracking and associated drilling."

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[1] http://www.environment.gov.au/water/coal-and-coal-seam-gas/publications/overview-assessment

[2] https://www.environment.gov.au/water/publications/deeper-groundwater-hazard-screening-chemicals-used-in-csg

[3] Kassotis et al (2013) Estrogen and Androgen Receptor Activities of Hydraulic Fracturing Chemicals and Surface and Ground Water in a Drilling-Dense Region, *Endocrinology* http://www.endo.endojournals.org

[4] Webb et al (2014) Unconventional oil and gas operations: developmental and reproductive effects. *Rev Environ Health* 2014; 29(4): 307–318

[5] Epigenetics refers to heritable changes in gene expression (active versus inactive genes) that does not involve changes to the underlying DNA sequence (source: http://www.whatisepigenetics.com/fundamentals/)

[6] Webb et al 2014

[7] Kassotis et al (2013) Estrogen and Androgen Receptor Activities of Hydraulic Fracturing Chemicals and Surface and Ground Water in a Drilling-Dense Region, *Endocrinology* doi: 10.1210/en.2013-1697 http://www.endo.endojournals.org

Dr Lloyd-Smith's troubling synopsis of the failures inherent in the NICNAS report is entirely in keeping with a recent paper published by Dr Linda Birnbaum, "Regulating toxic chemicals for public and environmental health" where she documents the lack of safety data and the ongoing failures of the regulators with regard to industrial chemicals in our environment. I attach Dr Birnbaum's paper as part of my submission.

It would seem, at best, that NICNAS's desktop assessment is 6 years out of date, (ie up to 2012) and they have not evaluated the replacement chemicals used by the industry.

In light of the fact that we know the risk of surface spills in the gas industry is high, and experience with PFOS/PFOA has shown that aquifers have been contaminated by surface spills, NICNAS'S dismissal of the risks is inexplicable.

It is inexplicable also that NICNAS could come to the conclusion that the identified exposure pathways were either unlikely or extremely unlikely despite the report noting that there was no information on well failure rates in Australia.

I would suggest to the panel that NICNAS's patently defective assessment of fracking chemicals simply reinforces the fact that the risks people of the NT will be exposed to if hydraulic fracturing goes ahead are significant and inadequately assessed.

3. Hospitalisation in Queensland

Since my meeting with members of the Inquiry Panel, my peer reviewed paper "Air Pollution and human health hazards: a compilation of air toxins acknowledged by the gas industry in Queensland's Darling Downs" has been published in the International Journal of Environmental Studies.
In it I document the startling rates of acute hospitalisation of Darling Downs residents. Between the years of 2007 and 2014 hospitalisation of residents of the Darling Downs for acute respiratory conditions more than doubled. Hospitalisation of Darling Downs' residents with acute circulatory conditions also more than doubled. This was also the time frame when the CSG industry was ramping up their activities in the Darling Downs with a resulting escalation of acknowledged air toxins. (I attach the paper as part of my submission).

I would ask the members of the panel to take on board these hospitalisation figures (which were supplied by the Darling Downs Hospital and Health Services), and in light of these figures evaluate the risk to the people of the NT.

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26th January 2018

PLOS BIOLOGY

EDITORIAL

Regulating toxic chemicals for public and environmental health

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This Editorial is part of the *Challenges in Environmental Health: Closing the Gap between Evidence and Regulations Collection*.

By the time President Gerald Ford signed the United States Toxic Substances Control Act in the fall of 1976, tens of thousands of synthetic chemicals had entered world markets with no evidence of their safety. Ford's signing statement described a law giving the Environmental Protection Agency (EPA) broad regulatory authority to require toxicity testing and reporting to determine whether the chemicals posed risks. "If a chemical is found to present a danger to health or the environment," Ford promised, "appropriate regulatory action can be taken before it is too late to undo the damage."

That's not what happened. The 60,000-plus chemicals already in commerce were grandfathered into the law on the assumption that they were safe. And the EPA faced numerous hurdles, including pushback from the chemical industry, that undermined its ability to implement the law. Congress finally revised the law last year, with the Frank R. Lautenberg Chemical Safety for the 21st Century Act, to bolster the EPA's regulatory authority. Over the decades that US policy on chemicals stagnated, scientists documented the damage whole classes of chemicals inflicted on living organisms and the environment that sustains them. Although we still have safety data on just a fraction of the 85,000-plus chemicals now approved for use in commerce, we know from field, wildlife, and epidemiology studies that exposures to environmental chemicals are ubiquitous. Hazardous chemicals enter the environment from the factories where they're made and added to a dizzying array of consumer products—including mattresses, computers, cookware, and plastic baby cups to name a few—and from landfills overflowing with our cast-offs. They drift into homes from nearby agricultural fields and taint our drinking water and food. Today, hundreds of industrial chemicals contaminate the blood and urine of nearly every person tested, in the US and beyond.

In the decades since Ford promised a robust policy to regulate potentially hazardous chemicals, evidence has emerged that chemicals in widespread use can cause cancer and other chronic diseases, damage reproductive systems, and harm developing brains at low levels of exposure once believed to be harmless. Such exposures pose unique risks to children at critical windows of development—risks that existing regulations fail to consider. To address these issues, *PLOS Biology* is publishing a special collection of seven articles, Challenges in Environmental Health: Closing the Gap between Evidence and Regulations, that focus on US chemical policy [1].

In commissioning the collection, we aimed to reveal barriers to developing health-protective policies not only when the scientific evidence of harm is clear but also when it is uncertain. We sought to explore the technical challenges involved in determining how the hundreds of

Citation: Gross L, Birnbaum LS (2017) Regulating toxic chemicals for public and environmental health. PLoS Biol 15(12): e2004814. https://doi.org/10.1371/journal.pbio.2004814

Published: December 18, 2017

Funding: The authors received no specific funding for this work.

Competing interests: I have read the journal's policy and the authors of this manuscript have the following competing interests. Liza Gross is a current paid employee at the Public Library of Science.

Provenance: Not commissioned; not externally peer reviewed.

chemicals we carry in our bodies affect health. These challenges include ascertaining exposures and impacts of short-lived compounds; identifying chemicals that pose unique risks to the developing fetus; and assessing the risk of chemicals that cause proportionately more harm at the lowest levels of exposure in violation of longstanding toxicology principles. We asked authors to consider these issues within their field of expertise and to suggest ways to bridge the gap between evidence and policy.

Several articles explore the failure of regulations to keep hazardous chemicals from polluting our food, air, and drinking water. Maricel Maffini and her colleagues describe the failure of regulators to account for health risks associated with the thousands of chemicals introduced into the food system since 1958, when Congress authorized the Food and Drug Administration to ensure the safety of substances added to food [2]. Sheldon Krimsky argues that an "unreasonable risk" standard to assess industrial chemicals in both the original and revised Toxic Substances Control Acts has imposed enormous data gathering and resource demands on the EPA, and ultimately hobbled the agency's ability to regulate [3].

But as Bruce Lanphear points out, no policy will protect public health if it doesn't account for the upending of one of toxicology's most fundamental precepts: the dose makes the poison [4]. Over the past three decades, Lanphear notes, evidence from some of the most extensively studied toxic chemicals—including lead, asbestos, tobacco, and benzene—shows that some chemicals are most toxic at the lowest levels of exposure. Yet regulations still assume that toxic effects emerge at a threshold level and increase with the dose. Protecting public health, Lanphear argues, requires rethinking basic assumptions about how agencies regulate chemicals.

Existing policy also fails to account for the fact that individuals are exposed to multiple chemicals every day, from the point of conception to the end of life. As Joseph Braun and Kimberly Gray note, epidemiologists are working to determine the full range of chemicals we carry in our bodies and how they affect health [5]. Toward that end, they're developing new methods to accurately estimate exposure to chemical mixtures, identify periods of heightened vulnerability, and flag chemicals that are particularly hazardous to children's health.

But having solid scientific evidence that a chemical causes harm, even to our children, is no guarantee that policymakers will act accordingly, Leo Trasande argues [6]. Using the failure to ban the pesticide chlorpyrifos as a case study, Trasande lays out the evidence that organophosphate pesticides like chlorpyrifos can damage the developing brain and impair cognitive and behavioral function through multiple mechanisms. The EPA reviewed this evidence and proposed a ban on chlorpyrifos in 2015, citing potential risks posed to women, children, and agricultural communities and workers [7]. The Trump administration reversed the ban earlier this year under "false scientific pretenses," Trasande argues. He calls on scientists to decry such attacks on human health and scientific integrity.

In the absence of a ban on chemicals known to cause harm, one option includes limiting their use around the most vulnerable populations. In California, state officials proposed limiting applications of agricultural pesticides within a quarter of a kilometer of schools and childcare centers after health officials reported that high levels of the chemicals were used near schools. The proposed buffer zone is a step in the right direction, argue Robert Gunier and his colleagues [8]. But a policy designed to safeguard vulnerable populations must account for additive effects of chemical mixtures, the different properties of the wide range of pesticides used in agriculture, and the lack of data to show what distance is truly protective. "The ideal solution to protecting children and pregnant women is an overall reduction in the use of agricultural pesticides to reduce exposure at home and at work, as well as at school," the authors argue.

Chemicals from agriculture, industry, and other commercial uses routinely enter drinking water supplies. One class of chemicals detected in drinking water, called perfluoroalkyl acids

(PFAAs), has come under increased scrutiny because of rapidly emerging evidence that these persistent chemicals accumulate in tissues and cause numerous adverse health effects, even at low levels. Recent research indicates that blood levels of these compounds increase on average by more than 100 times their concentration in drinking water, note Gloria Post and her colleagues [9]. Drinking water guidelines must account for the fact that infants receive much higher exposures than adults from the same drinking water source, and retain these compounds in their bodies years after exposure ends, the authors argue.

As the contributors to this special collection make clear, existing US regulations have not kept pace with scientific advances showing that widely used chemicals cause serious health problems at levels previously assumed to be safe. The most vulnerable population, our children, face the highest risks. More research is needed to better understand the risks posed by these chemicals, identify susceptible groups, and develop safe alternatives. But as the contributors also make clear, science is not always enough. Closing the gap between evidence and policy will require that engaged citizens, both scientists and nonscientists, work to ensure our government officials pass health-protective policies based on the best available scientific evidence.

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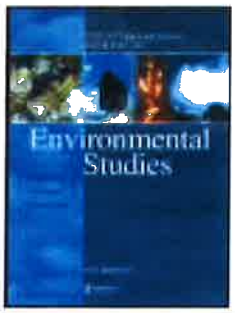

International Journal of Environmental Studies

ISSN: 0020-7233 (Print) 1029-0400 (Online) Journal homepage: http://www.tandfonline.com/loi/genv20

Air Pollution and human health hazards: a compilation of air toxins acknowledged by the gas industry in Queensland's Darling Downs

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To cite this article: Geralyn McCarron (2018): Air Pollution and human health hazards: a compilation of air toxins acknowledged by the gas industry in Queensland's Darling Downs, International Journal of Environmental Studies, DOI: 10.1080/00207233.2017.1413221

To link to this article: https://doi.org/10.1080/00207233.2017.1413221

INTERNATIONAL JOURNAL OF ENVIRONMENTAL STUDIES, 2018
https://doi.org/10.1080/00207233.2017.1413221

Routledge
Taylor & Francis Group

Air Pollution and human health hazards: a compilation of air toxins acknowledged by the gas industry in Queensland's Darling Downs

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ABSTRACT

The paper offers an attempt to determine whether emissions from the unconventional gas industry are associated with hospitalisations in the Darling Downs, Queensland, Australia. Hospitalisation data were obtained from the Darling Downs Hospital and Health Services (DDHHS) and Coal Seam Gas (CSG) emissions data from the National Pollutants Inventory (NPI). Hospital admissions for circulatory and respiratory conditions, controlled for population, increased significantly from 2007 to 2014 ($p < 0.001$). Acute circulatory admissions increased 133% (2198–5141) and acute respiratory admissions increased 142% (1257–3051). CSG emissions increased substantially over the same period: nitrogen oxides (489% to 10,048 tonnes), carbon monoxide (800% to 6800 tonnes), PM10 (6000% to 1926 tonnes), volatile organic compounds (337% to 670 tonnes) and formaldehyde (12 kg to over 160 tonnes). Increased cardiopulmonary hospitalisations are coincident with the rise in pollutants known to cause such symptoms. Apparently, controls to limit exposure are ineffectual. The burden of air pollution from the gas industry on the wellbeing of the Darling Downs population is a significant public health concern.

KEYWORDS

Queensland; unconventional; gas; emissions; cardiorespiratory; hospitalisation

Introduction

The Darling Downs (Figure 1) west of the Great Dividing Range in Southern Queensland, Australia has long been noted for its robust, diversified agricultural industry and natural beauty [1]. The Darling Downs Hospital and Health Service (DDHHS) covers an area of approximately 90,000 sq km with catchment population ca. 277,000 [2]. There has been rapid development of the resources industry (CSG, underground gasification, coal), superimposed on pre-existing rural, farming and small town communities in the area now often known by its geological name, 'the Surat Basin'.

Outdoor air pollution, especially in an industrial context, has demonstrated multiple negative human health effects [3]. Air pollution increases risks for a wide range of diseases including respiratory [4] and cardiac [5,6], and is a leading environmental

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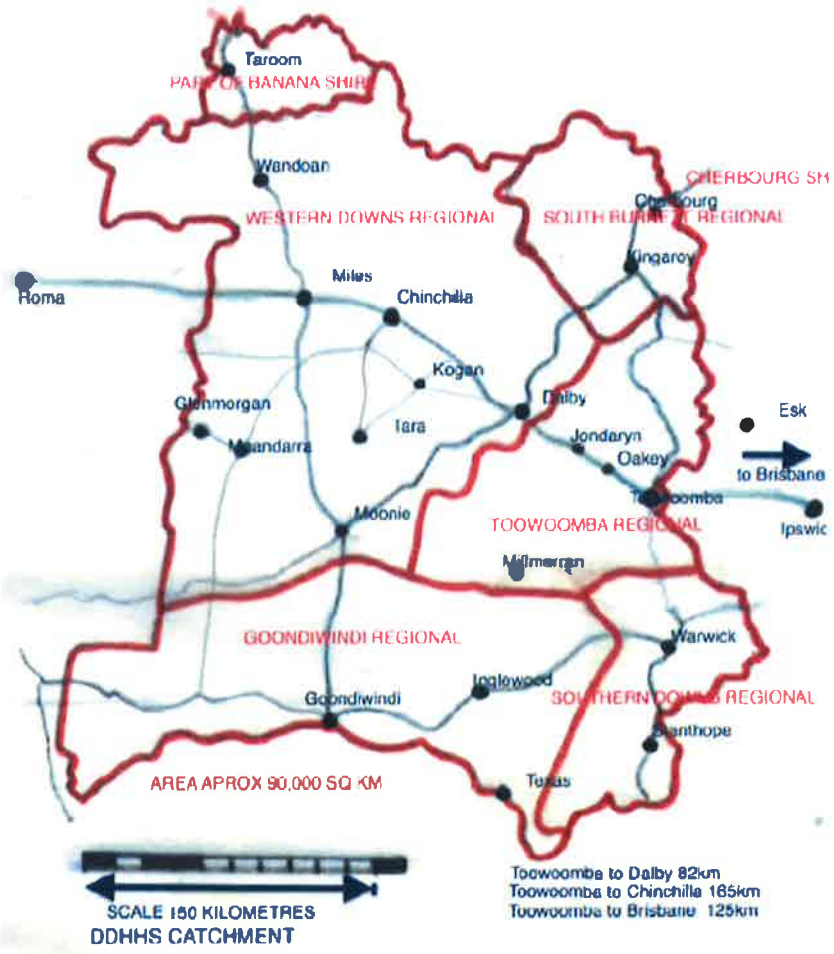

Figure 1. Map DDHHS catchment showing Local Government Areas. Source: Author.

cause of cancer deaths [7]. Some effects are long-term and causation can be difficult to prove. For instance, a heart attack or stroke resulting from exposure during a day of high ambient PM concentration may be a consequence of chronic disease progression associated with long-term exposure [8]. Emissions acknowledged by the CSG industry can be linked to both acute and chronic health effects (see Table 1, summary of air toxins/related health effects).

The unchecked expansion of unconventional gas companies into what was previously an agrarian area of the Darling Downs has led to the generation of extra emissions attributable to a single industry.

Table 1. Air toxins and associated health effects.

Air toxin	Health effect
Oxides of Nitrogen (NOx) including Nitrogen dioxide (NO_2)	• An irritant asphyxiant dissolving on moist tissue surfaces to form nitric acid, irritates and burns delicate tissues. • Acute health effects [9] include eye, throat and lung irritation, wheezing and tight chest [10]. Triggers asthma. • Chronic health effects of NO_2 exposure include an *'asthma–like condition called RADS'*, as well as *'obliterative bronchiolitis'* [11]. • Associated with deficits in children's lung function growth [12]. • Exposure significantly associated with acute emergency visits [13], hospitalization for asthma and all respiratory diagnoses [14].
Carbon monoxide (CO)	• Chemical asphyxiant, People with pre-existing ischaemic heart disease are the most sensitive group for CO exposure at ambient/near ambient concentrations, significantly increasing arrhythmias and angina [15].
Particulate Matter PM_{10} and $PM_{2.5}$	• $PM_{2.5}$ is a cause of cardiovascular mortality and morbidity [3]. • Linked to childhood respiratory disease [16,17], atherosclerosis, and adverse birth outcomes. • Coarse PM [18] has at least as strong short-term effects on respiratory health as $PM_{2.5}$. • Possible links to chronic disease conditions: diabetes, neurodevelopment and cognitive function.
Volatile Organic Compounds (VOCs)	• Irritation to the eyes, nose and throat; • Headaches; incoordination; nausea; liver, kidney and central nervous system damage. • Some VOCs are known human carcinogens.
Ozone (formed when NO_2 and VOCs react together in presence of sunlight.	• Exposure results in airway inflammation, airway hyper-responsiveness, and decrements in lung function in healthy and asthmatic adults [3]. • Asthma admissions associated significantly with 3 indicators of chronic ozone exposure (mean concentration, summer mean and percentage days with ozone levels greater than 35 ppb) [19].
Sulphur Dioxide (SO_2)	• Irritation of the eyes, nose, throat, and airways causing inflammation, wheezing and lung damage [20]. • Positive and statistically significant with respiratory hospital admissions [3]. • Those with impaired heart or lung function at increased risk.
Formaldehyde	• Documented sensory irritant, causing burning sensations in the eyes, nose, and throat, coughing and wheezing [21]. • Implicated in worsening of allergic and respiratory symptoms in children. • Known human carcinogen [22] linked to nasopharyngeal cancer, sinonasal cancer and lymphohaematopoietic cancer specifically myeloid leukaemia.

Concurrent with the rapidly expanding CSG developments, residents in Queensland's Darling Downs reported impairments to their health [23,24]. As acknowledged by the Darling Downs Public Health Unit (DDPHU) health impacts associated with Coal Seam Gas have been a major community concern. Since 2008 DDPHU has received a variety of health complaints related to this industry (including headaches, sore eyes, nosebleeds, rashes, respiratory symptoms, paraesthesia) [25].

Yet there has been a remarkable lack of substantive investigation into potential human health impacts of the CSG industry in the Darling Downs. No baseline environmental studies, human health risk assessments or health studies were undertaken before large-scale extraction took place. State-based research organizations expected to be active in the space have disclosed little research investigating the possible physical health impacts of unconventional gas emissions. The significant 2010 Australian Research Council linkage project 'A Human Health Risk Assessment for developing CSG water resources in Queensland' [26] was not pursued, purportedly because the industry partner, Santos, withdrew funding. A notable exception is the work of Werner et al. [27] reviewing hospitalisation data up to 2011 for 3 areas in Queensland, with the finding that certain hospital admissions rates (neoplasms and blood/immune diseases) increased more quickly in the CSG area than the other study areas, after adjusting for key sociodemographic factors. In other jurisdictions, specifically the USA, increased rate and severity of asthma attacks [28], increased hospitalisation [29] for asthma, cardiac, neurological and skin conditions, increased incidence of congenital heart defects [30], childhood leukaemia [31], low birth weight [32], and early infant death [33] correlated with the presence of the unconventional gas industry. International researchers have documented significant declines in air quality correlating with gas industry activities [34–36].

Despite appeals from health professionals to improve oversight, state and federal regulatory bodies have failed to act. In 2013 the Australian Medical Association (AMA) issued a policy statement warning: 'Despite the rapid expansion of CSG developments, the health impacts have not been adequately researched, and effective regulations that protect public health are not in place' [37]. In 2013 also, the Queensland Government undertook a limited investigation into health complaints of Darling Downs residents [38]. The report, while unable to determine whether reported health effects were clearly linked to exposure to CSG pollutants, acknowledged that there was 'some evidence that might associate some of the residents' symptoms to exposure to airborne contaminants arising from CSG activities.' The critical recommendation from Queensland Health was that the regulator, the Department of the Environment and Heritage Protection (DEHP) monitor overall CSG emissions and the exposure of local communities to those emissions. DEHP acknowledged that they did not have access to data to allow for comparisons to the air quality objectives set out in the Environmental Protection Policy (EPP) (Air) to protect environmental values (including health and wellbeing). Despite this, DEHP determined that they found no cause to expand monitoring, thereby blocking Queensland Health's recommendation that overall gasfield emissions and the exposure of the community to those emissions be monitored [39]. The rejection by the regulator of these recommendations is of serious concern.

The anecdotal reports of health effects related to CSG industry activity [23], coupled with the dearth of available research in the Australian context, motivated this investigation. This paper seeks to compile available reported emissions from CSG installations in the Darling Downs area and determine whether such activity is coincident with an increase in

acute health effects. It brings together data on air pollutants as reported by the industry to the National Pollutant Inventory (NPI) [40], population data from the Australian Bureau of Statistics (ABS) and hospitalisation data from the Darling Downs Hospital and Health Services (DDHHS).

Method

Population data

Estimated resident population by local government area (LGA), Queensland, 2007–2014 (Queensland Government Statistician's Office) was sourced from ABS 3218.0, Regional Population Growth, Australia, 2015–2016 and Queensland Treasury estimates where Geographies were based on the 2016 edition of the Australian Statistical Geography Standard (ASGS) [41].

Air toxics data

Reported emissions to air were obtained from the Commonwealth Department of the Environment's National Pollutant Inventory Website [40]. Above a defined threshold, the Australian Government requires polluting industries to self-report to the NPI their calculated, estimated emissions of 93 toxic substances, identified as important owing to their possible effect on human health and the environment.

Hospital admission data

Inpatient admissions were obtained for DDHHS and South West residents treated within all acute public hospital facilities interfaced to the Transition 11 clinical benchmarking system. This included all Activity Based Funding (ABF) facilities and the majority of satellite facilities within each HHS, but excluded the Brisbane Mater Adult, Mothers and Children's Hospitals. Hospitalisation of residents from South West has been separated from the main DDHHS data, as with such a large, sparsely populated area (310,000 km^2, population approximately 26,000) it is difficult to draw inferences regarding activity and effects. Data on hospital admissions were provided by DDHHS for the years 2006–2015. Data for 2006 were partial (01/07/06 to 31/12/06) and data for 2015 was also incomplete (01/01/15 to 18/08/15) and were excluded.

Statistical analysis

Acute hospital admission data (circulatory and respiratory) were controlled for population increases in the DDHHS catchment. Linear regression analysis was performed using SPSS.

Results

In the DDHHS region, in 2015 there were 3521 registered births and 2353 registered deaths. In 2011, 2.4% of the population (2763) in the region was employed in mining.[1] Statistics from the ABS indicate population increase from 252,785 to 276,723 [41]. The region with

Table 2. DDHHS acute hospital admissions by residence and year.

	2007	2008	2009	2010	2011	2012	2013	2014
Circulatory								
Chinchilla	65	53	63	57	167	289	372	331
Dalby	89	97	82	88	262	516	531	503
Goondiwindi	33	23	32	27	102	216	164	164
Inglewood	7	16	6	11	21	33	56	49
Kingaroy	86	109	93	102	194	374	342	419
Miles	18	35	28	22	76	123	112	94
Millmerran	20	20	17	25	41	64	75	58
Stanthorpe	50	63	61	69	203	318	336	315
Texas	11	12	16	11	28	40	43	36
Toowoomba	1485	1736	1691	1834	2023	2159	2391	2606
Warwick	334	536	533	571	629	641	614	566
Total	2198	2700	2622	2817	3746	4773	5036	5141
Respiratory								
Chinchilla	24	27	41	38	112	206	195	194
Dalby	46	61	64	55	211	308	316	291
Goondiwindi	12	8	14	12	101	142	141	134
Inglewood	1	1	6	4	29	53	50	28
Kingaroy	46	49	61	55	156	213	237	243
Miles	9	13	12	12	47	75	78	70
Millmerran	5	11	7	13	23	50	42	32
Stanthorpe	17	25	33	36	122	147	145	174
Texas	3	7	10	5	12	31	22	26
Toowoomba	992	1035	1032	1152	1224	1317	1278	1513
Warwick	172	312	284	315	373	321	287	346
Total	1257	1549	1564	1697	2410	2863	2781	3051
DDHHS catchment population[a]	252,785	256,824	261,109	264,185	267,052	270,851	274,536	276,723

[a]Population DDHHS catchment based on local government areas Toowoomba, Western Downs, Southern Downs, South Burnett, Cherbourg, Goondiwindi and the Taroom community of Banana Shire.

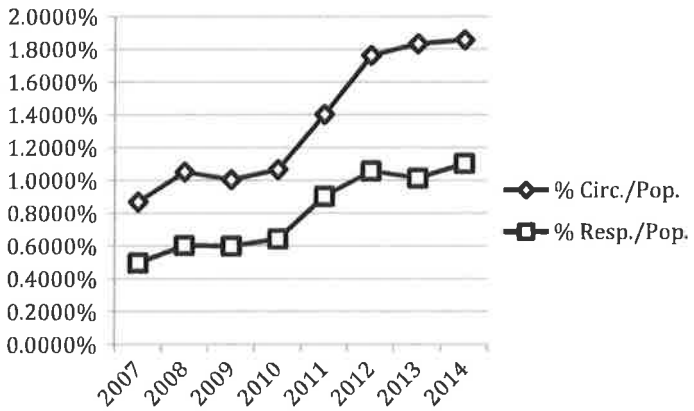

Figure 2. DDHHS acute circulatory and respiratory hospital admissions as a percentage of population from 2007 to 2014.

regard to Queensland regional statistics means the area serviced by the Darling Downs Hospital and Health Service as shown by their maps; including Cherbourg, Goondiwindi, South Burnett, Southern Downs, Toowoomba and Western Downs, and Taroom.[2,3]

Linear regression analysis was performed on the hospital admissions data, controlled for population, versus time (Table 2 and Figure 2). Admissions for

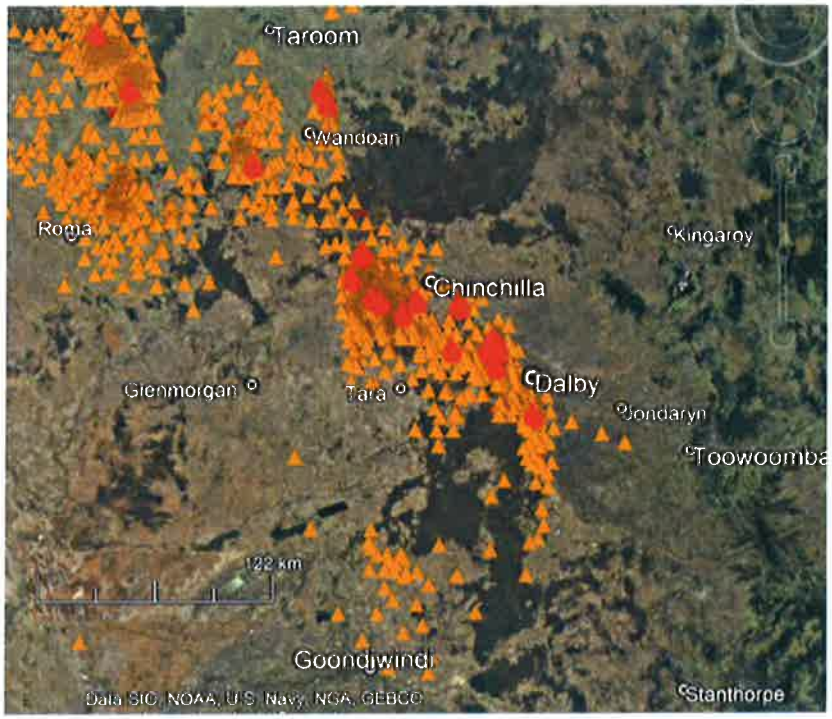

Figure 3. Map: gas wells (triangles), CSG emission reporting sites (flame). Source Google Earth Pro, overlay Landstat/Copernicus. (cited 2017 August 4).

Table 3. Compilation NPI data, self-reported emissions (kilograms) QGC, Origin, Santos, Arrow facilities DDHHS Western Darling Downs catchment 2005/06–2015/16.

KG year	Carbon monoxide	Oxides of nitrogen	Total VOCs	PM10	PM2.5	formalde-hyde	Sulphur dioxide
2005/06	143,200	952,700	94,400	11,200	0	0	1148
2006/07	754,000	1,704,000	153,400	29,210	0	12	1143
2007/08	1,208,000	2,243,300	838,100	33,350	1,210	25	1061
2008/09	3,684,000	7,258,000	438,500	17,994	17,664.1	14,700	2192
2009/10	1,064,600	2,877,000	632,420	35,455	12,773.2	0	3823
2010/11	2,273,600	7,218,200	991,200	116,105	94,052	85,000	10,442
2011/12	2,447,500	8,705,000	947,000	164,170	121,179	119,000	11,130
2012/13	2,523,000	6,477,000	762,600	2,051,207	172,926.6	150,000	11,074
2013/14	6,800,000	10,048,000	670,600	1,926,907	301,113.8	160,420	12,976
2014/15	8,719,000	11,584,000	887,900	5,572,422	252,939	254,200	16,692
2015/16	6,473,000	10,947,000	2,640,130	4,621,514	187,533.1	307,200	15,704

circulatory conditions significantly increased over the period from 0.87% in 2007 to 1.86% in 2014 ($R^2 = 0.908$, $p < 0.001$). Respiratory admissions also significantly increased from 0.50% in 2007 to 1.10% in 2014 ($R^2 = 0.913$, $p < 0.001$).

Figure 3 shows a map of the Darling Downs showing towns, gas wells and gas facilities. Table 3 is a compilation of emissions reported to the NPI by QGC, Origin, Santos, and Arrow facilities in the DDHHS catchment (2005/06–2015/16). Figures 4 and 5 graph selected emissions (2007–2014).

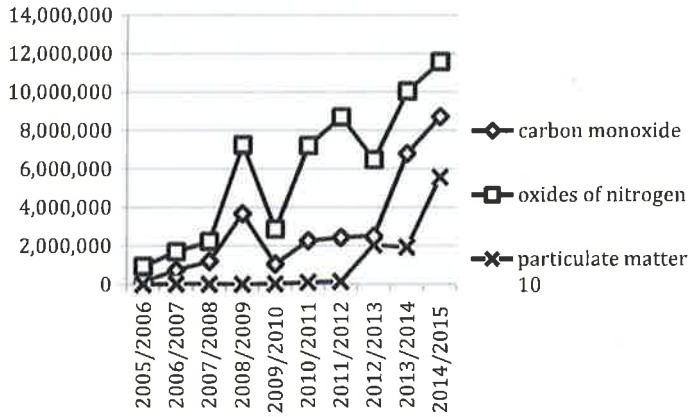

Figure 4. Selected emissions: carbon monoxide, oxides of nitrogen, particulate matter 10, (Kilograms) Western Darling Downs, reported by CSG companies QGC, Origin, Arrow, Santos.

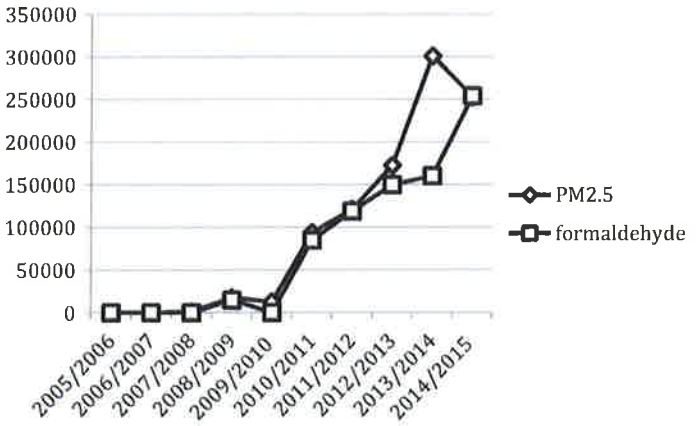

Figure 5. Selected emissions: Particulate matter 2.5, formaldehyde (Kilograms) Western Darling Downs, reported by CSG companies QGC, Origin, Arrow, Santos.

Limitations

There are limitations to the data available for consideration. For instance it was not possible to break down admissions into demographics (age/gender) and times/dates of admission. Factors for which data are unavailable are the change, if any, in the population rates of cigarette smoking and obesity, and the prior health status of residents who may have moved into (and out of) the area between 2007 and 2014. The contribution to ill health of viral epidemics is unknown (e.g. swine flu 2009, but the expected peak and return to baseline in the following year did not occur (Table 2).

NPI reporting of emissions is annual, with no data available on times/dates/durations of peak emissions, and no data available on the prevailing weather conditions. The difficulty in correlating lower volume pollutants to chronic (cancer, neurodevelopmental) health conditions is recognised, hence the need to rely on high volume pollutants and acute health effects. An assessment of the cumulative load of air toxins such as phenol, n-hexane, PAHs

and heavy metals was impossible as reporting was inconsistent. Other resource developments (coal/underground gasification), and possible changes in agricultural practices have not been studied.

It is also notable that the gross emission values provided by the NPI are industries' estimates of their releases to air. As these are total annual estimates, it is impossible to calculate the resulting concentration of pollutants, either individually or combined in the airshed at any given time with any confidence. Nor can the results be used to compare air emissions against any relevant national or state air quality guidelines or standards, given as concentrations. Estimates of the aggregate pollutant load and concentration do not capture spatial or temporal conditions, which may lead to dangerous exposure and therefore acute or chronic health effects (point emission, low air temperature, etc. See below).

Discussion

There are noted anomalies in the industry NPI data. In 2008/2009 across all Arrow projects, there was detailed reporting of a wide range of toxins, yet many were not reported previously or since. During that year, levels of carbon monoxide and oxides of nitrogen were significantly higher than recorded in the preceding/following year. No explanation is apparent. After 2009/2010 several projects across the Darling Downs and South West no longer reported benzene, though previously reporting significant volumes. Despite undertaking comparable activities, Santos consistently failed to report formaldehyde emissions while QGC reported up to 219 tonnes per year. Although reporting is a statutory requirement, data are self-calculated (estimated, not measured) and are not reported below a threshold. It is difficult to know how such reporting could be audited. It is plausible that emissions have been substantially underestimated.

Emissions reported by the CSG industry to the NPI have escalated since expansion of CSG from 2006 onwards. Toxins include particulate matter *with over 6,000% increase* in reported emissions of PM_{10} between the years of 2006/2007 and 2013/2014 (29.19–1926.9 tonnes). Reported emissions of $PM_{2.5}$ increased from zero to 301 tonnes. Emissions of oxides of nitrogen *increased by 489%,* (1704–10048 tonnes) VOCs by 337%, (153.4–670.6 tonnes) CO *by 801%,* (754–6,800 tonnes) SO_2 by *more than 1000%* (1.14–12.97 tonnes) and, remarkably emissions of the known carcinogen formaldehyde *increased from 12 kg to 160.42 tonnes* over the same time period. Further escalation in emissions is noted in the reporting periods 2014/2015, and 2015/2016 (Table 3).

Between the years of 2007 and 2014, hospitalisations of DDHHS patients for respiratory conditions increased by 142%, and hospitalisations for circulatory conditions increased by 133%. Hospitalisations from DDHHS areas fluctuated between 2007 and 2010 with significant rates of change apparent in 2010/2011 (circulatory conditions increased 32%, respiratory conditions 42%) and 2011/2012 (circulatory conditions increased 27%, respiratory conditions 18%). Increases were evident across all DDHHS areas including areas relatively distant from intense gas field industrialisation such as Goondiwindi and Inglewood. Interpretation of individual changes in these very low population centres is made more difficult by the number of drive in/drive out gasfield workers from across the Darling Downs.

Changes are not explicable by the modest population increase of 9.46% during the same time period, or the change in median age, which over the longer time frame of 2005–2015 increased by 2.4 years. They do however give weight to the community's perception that

there has been an adverse change in their health status. It is noted that these changes were not commented on in the Queensland Government 2013 CSG investigation. This may relate to the limits of their terms of reference and/or lag time with data compilation.

There is international acknowledgement of the serious adverse impacts on human health of air pollution in general, and the toxicity of the specific air pollutants reported by the gas industry to the NPI (see Table 1, summary of air toxins/related health effects). Many families, including young children are, for up to 24 h a day, living, breathing and sleeping in the midst of point emission sources in Queensland's gasfields. They are exposed to acute peaks and chronic, lower concentrations of mixtures of harmful chemicals. Air dispersion throughout the regional airshed means the broader population is likely to be repeatedly exposed to lower doses of the same toxins.

Although the quantity of emitted pollutants is notionally 'within guidelines,' there does not appear to be a level of emissions unacceptable to industry or the regulator. Former Queensland Premier Campbell Newman stated: 'Emission limits are not prescribed for each gas well or the broader reticulation system but rather, emissions from this infrastructure must not cause nuisance or environmental harm' (Letter from The Hon. Campbell Newman, Premier of Queensland, to Dr McCarron, 7th November 2012).

Currently, production facilities act with the assumption that emitted pollutants will be dispersed in the surrounding airshed to 'safe' levels (Dilution is assumed to be the solution to pollution) [42].

Since there is an unexplained rise in hospitalisations for health conditions associated with exposure to CSG emissions coincident with the expansion of the industry, it is questionable whether this management strategy is effective. Such a method for the neutralisation of harmful wastes largely ignores local environmental effects: large-volume point emissions, wind strength/direction and day/night temperature differences which could lead to adverse levels of exposure.

Australia has National Air Quality Standards with defined maximum limits for the aforementioned pollutants (CO, NO_2, Ozone, SO_2, Lead, PM_{10}) over specified averaging periods [43]. Yet without real-time 24 h monitoring, there is no way to know whether such standards have been exceeded. Additionally, deleterious health effects have been noted to occur at levels below current air quality guidelines, and for many pollutants it is not clear whether a safe threshold exists [3]. The Queensland Government has an ambient air-monitoring network [44], but before February 2015 there was not a single air monitoring station sited in the expanding gas fields, with no station west of Jondaryan (see Figure 3, operated March 2014–Aug 2016). Air monitoring has been infrequent, ad hoc, episodic and reactive [45]. Often, air monitoring did not occur until weeks after the local community reported extreme pollution events such as intense flaring. Monitoring and reporting practices for air quality appear inadequate to protect public health [46]. Drinkwater (2015) noted the limited monitoring data received through the RTI process, and queried whether there is a shortfall in the process or whether monitoring data simply do not exist [47]. Both considerations point to regulatory failure.

In this study the limitations of reporting requirements to the NPI were such that it was not possible to calculate the cumulative load of low volume highly toxic pollutants (phenol, PAH, BETX, heavy metals, etc.). Nevertheless, the need to monitor and restrict emissions of such pollutants is critically important, as they may be associated with future chronic health conditions, including cancer [48] and neurodevelopmental abnormalities [49].

DDHHS hospitalisation data for acute respiratory and circulatory conditions appear consistent with short-term health impacts of air pollution. Of concern is the future health of a population subject to chronic exposure. Long-term, real-time 24-h exposure monitoring to capture the temporal and spatial variability of a wide range of key environmental toxins *is necessary to assess exposure.* Average ambient levels do not give an adequate assessment of the health risks to vulnerable subgroups of the population [50]. This applies particularly to children, pregnant women, the ill, including those with pre-existing cardiac and respiratory disease, and the elderly. It applies to those living in close proximity to infrastructure, who are exposed to spikes of multiple air toxins, with increased risks on still nights during temperature inversions. These are the populations also at most risk to high volume pollutants. Children, with their high ventilation rates per body weight and increased activity and play outdoors, are particularly susceptible to the adverse effects of air pollution [51]. When exposed to mixtures of toxic chemicals they have heightened risk because of the immaturity of their immune and metabolic responses and their potential to live long enough for latent illnesses to develop [52]. It is increasingly recognised that even current air standards properly applied provide suboptimal protection for the most vulnerable in our society. Monitoring by the regulatory bodies has been ineffectual and inadequate to protect public health. Over the past decade, an unmistakeable and significant change in the life of residents of the Darling Downs has related to the arrival and activities of the CSG industry. There has been an escalation in health impairment correlating with, and potentially attributable to, the escalating air pollution from this heavy industry. This is demonstrated by acute respiratory and circulatory hospitalisation. Hospitalisation is an extreme indicator of morbidly and does not take into account the potential full spectrum of health harms experienced by the community.

Conclusion

Health impacts from Coal Seam Gas have been a major community concern since the introduction of CSG industries in Queensland. For almost a decade the community has recognised and reported concerns about their changed health status.

Whilst the full range of factors underlying the escalating hospitalisation of Darling Downs' residents for acute respiratory and circulatory conditions is unknown, the DDHHS statistics are significant and warrant full investigation as to causal factors. Communities in the Darling Downs have been exposed to significant pollution associated with the rapid and extreme industrialisation by the gas industry and with toxins directly attributable to that industry. The considerable growth in hospitalisations for acute respiratory and circulatory conditions concurrent with the increase in toxic pollutants in the local airspace suggests that controls to limit exposure are ineffectual.

A growing body of published research on the industry's emissions and resultant adverse health impacts supports the decisions by other jurisdictions (France, Ireland, Bulgaria, New York State), to impose bans on unconventional gas development. Acute hospitalisation data from the Darling Downs raise a red flag. It is urgent that there should be a comprehensive investigation of the health impacts from the unconventional gas industry in Australia.

Notes

1. 'Mining' includes coal mining, oil and gas extraction, metal ore mining, non-metallic mineral mining and quarrying, exploration and other mining support services.
2. http://www.qgso.qld.gov.au/index.php.
3. For reasons of space, the author is not including supplementary material in the paper, but it can be provided upon request.

Acknowledgements

Acknowledgement and thanks to the Darling Downs Hospital and Health Service for providing data relating to hospital admissions. The author is grateful to Dr Rye Senjen, Dr Jed Burns and Ms Shay Dougall for review, comments and assistance with statistics and maps.

Disclosure statement

No potential conflict of interest was reported by the author.

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PLOS BIOLOGY

EDITORIAL

Regulating toxic chemicals for public and environmental health

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This Editorial is part of the *Challenges in Environmental Health: Closing the Gap between Evidence and Regulations Collection*.

By the time President Gerald Ford signed the United States Toxic Substances Control Act in the fall of 1976, tens of thousands of synthetic chemicals had entered world markets with no evidence of their safety. Ford's signing statement described a law giving the Environmental Protection Agency (EPA) broad regulatory authority to require toxicity testing and reporting to determine whether the chemicals posed risks. "If a chemical is found to present a danger to health or the environment," Ford promised, "appropriate regulatory action can be taken before it is too late to undo the damage."

That's not what happened. The 60,000-plus chemicals already in commerce were grandfathered into the law on the assumption that they were safe. And the EPA faced numerous hurdles, including pushback from the chemical industry, that undermined its ability to implement the law. Congress finally revised the law last year, with the Frank R. Lautenberg Chemical Safety for the 21st Century Act, to bolster the EPA's regulatory authority. Over the decades that US policy on chemicals stagnated, scientists documented the damage whole classes of chemicals inflicted on living organisms and the environment that sustains them. Although we still have safety data on just a fraction of the 85,000-plus chemicals now approved for use in commerce, we know from field, wildlife, and epidemiology studies that exposures to environmental chemicals are ubiquitous. Hazardous chemicals enter the environment from the factories where they're made and added to a dizzying array of consumer products—including mattresses, computers, cookware, and plastic baby cups to name a few—and from landfills overflowing with our cast-offs. They drift into homes from nearby agricultural fields and taint our drinking water and food. Today, hundreds of industrial chemicals contaminate the blood and urine of nearly every person tested, in the US and beyond.

In the decades since Ford promised a robust policy to regulate potentially hazardous chemicals, evidence has emerged that chemicals in widespread use can cause cancer and other chronic diseases, damage reproductive systems, and harm developing brains at low levels of exposure once believed to be harmless. Such exposures pose unique risks to children at critical windows of development—risks that existing regulations fail to consider. To address these issues, *PLOS Biology* is publishing a special collection of seven articles, Challenges in Environmental Health: Closing the Gap between Evidence and Regulations, that focus on US chemical policy [1].

In commissioning the collection, we aimed to reveal barriers to developing health-protective policies not only when the scientific evidence of harm is clear but also when it is uncertain. We sought to explore the technical challenges involved in determining how the hundreds of

Citation: Gross L, Birnbaum LS (2017) Regulating toxic chemicals for public and environmental health. PLoS Biol 15(12): e2004814. https://doi.org/10.1371/journal.pbio.2004814

Published: December 18, 2017

Funding: The authors received no specific funding for this work.

Competing interests: I have read the journal's policy and the authors of this manuscript have the following competing interests. Liza Gross is a current paid employee at the Public Library of Science.

Provenance: Not commissioned; not externally peer reviewed.

chemicals we carry in our bodies affect health. These challenges include ascertaining exposures and impacts of short-lived compounds; identifying chemicals that pose unique risks to the developing fetus; and assessing the risk of chemicals that cause proportionately more harm at the lowest levels of exposure in violation of longstanding toxicology principles. We asked authors to consider these issues within their field of expertise and to suggest ways to bridge the gap between evidence and policy.

Several articles explore the failure of regulations to keep hazardous chemicals from polluting our food, air, and drinking water. Maricel Maffini and her colleagues describe the failure of regulators to account for health risks associated with the thousands of chemicals introduced into the food system since 1958, when Congress authorized the Food and Drug Administration to ensure the safety of substances added to food [2]. Sheldon Krimsky argues that an "unreasonable risk" standard to assess industrial chemicals in both the original and revised Toxic Substances Control Acts has imposed enormous data gathering and resource demands on the EPA, and ultimately hobbled the agency's ability to regulate [3].

But as Bruce Lanphear points out, no policy will protect public health if it doesn't account for the upending of one of toxicology's most fundamental precepts: the dose makes the poison [4]. Over the past three decades, Lanphear notes, evidence from some of the most extensively studied toxic chemicals—including lead, asbestos, tobacco, and benzene—shows that some chemicals are most toxic at the lowest levels of exposure. Yet regulations still assume that toxic effects emerge at a threshold level and increase with the dose. Protecting public health, Lanphear argues, requires rethinking basic assumptions about how agencies regulate chemicals.

Existing policy also fails to account for the fact that individuals are exposed to multiple chemicals every day, from the point of conception to the end of life. As Joseph Braun and Kimberly Gray note, epidemiologists are working to determine the full range of chemicals we carry in our bodies and how they affect health [5]. Toward that end, they're developing new methods to accurately estimate exposure to chemical mixtures, identify periods of heightened vulnerability, and flag chemicals that are particularly hazardous to children's health.

But having solid scientific evidence that a chemical causes harm, even to our children, is no guarantee that policymakers will act accordingly, Leo Trasande argues [6]. Using the failure to ban the pesticide chlorpyrifos as a case study, Trasande lays out the evidence that organophosphate pesticides like chlorpyrifos can damage the developing brain and impair cognitive and behavioral function through multiple mechanisms. The EPA reviewed this evidence and proposed a ban on chlorpyrifos in 2015, citing potential risks posed to women, children, and agricultural communities and workers [7]. The Trump administration reversed the ban earlier this year under "false scientific pretenses," Trasande argues. He calls on scientists to decry such attacks on human health and scientific integrity.

In the absence of a ban on chemicals known to cause harm, one option includes limiting their use around the most vulnerable populations. In California, state officials proposed limiting applications of agricultural pesticides within a quarter of a kilometer of schools and childcare centers after health officials reported that high levels of the chemicals were used near schools. The proposed buffer zone is a step in the right direction, argue Robert Gunier and his colleagues [8]. But a policy designed to safeguard vulnerable populations must account for additive effects of chemical mixtures, the different properties of the wide range of pesticides used in agriculture, and the lack of data to show what distance is truly protective. "The ideal solution to protecting children and pregnant women is an overall reduction in the use of agricultural pesticides to reduce exposure at home and at work, as well as at school," the authors argue.

Chemicals from agriculture, industry, and other commercial uses routinely enter drinking water supplies. One class of chemicals detected in drinking water, called perfluoroalkyl acids

(PFAAs), has come under increased scrutiny because of rapidly emerging evidence that these persistent chemicals accumulate in tissues and cause numerous adverse health effects, even at low levels. Recent research indicates that blood levels of these compounds increase on average by more than 100 times their concentration in drinking water, note Gloria Post and her colleagues [9]. Drinking water guidelines must account for the fact that infants receive much higher exposures than adults from the same drinking water source, and retain these compounds in their bodies years after exposure ends, the authors argue.

As the contributors to this special collection make clear, existing US regulations have not kept pace with scientific advances showing that widely used chemicals cause serious health problems at levels previously assumed to be safe. The most vulnerable population, our children, face the highest risks. More research is needed to better understand the risks posed by these chemicals, identify susceptible groups, and develop safe alternatives. But as the contributors also make clear, science is not always enough. Closing the gap between evidence and policy will require that engaged citizens, both scientists and nonscientists, work to ensure our government officials pass health-protective policies based on the best available scientific evidence.

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