**Ohio Senate**
**Committee on Energy and Public Utilities**
**Testimony on Senate Bill 315**

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Chairwoman Jones, Vice-Chairman Balderson, Ranking Member Schiavoni and members of the Senate Energy and Public Utilities Committee, thank you for the opportunity to offer testimony on Senate Bill 315 as introduced.

My name is Matt Watson. I serve as a Senior Energy Policy Manager at the Environmental Defense Fund. Since 1967 EDF has linked science, economics and law to create innovative, equitable and cost-effective solutions to urgent environmental problems. We have more than 700,000 members worldwide, including more than 32,000 in Ohio.

EDF is somewhat unique in that we seek market-based solutions to environmental problems, wherever it makes sense. And while we accept no corporate funding, we're known for our efforts to work with industry to achieve our environmental goals. As just one example of this, in partnership with Southwestern Energy – a major natural gas producer in the Fayetteville and Marcellus shales and elsewhere – we've brought together other producers and environmental groups to develop model rules for well construction and operation. That's an ongoing project. Yet, even though not yet complete, we are pleased that is has helped inform the well construction rulemaking that's nearing the finish line in Ohio.

Over the past two years we have helped shape the fracturing fluid chemical disclosure policies adopted by Arkansas, Texas, Montana, Colorado, Pennsylvania and those awaiting final approval in Oklahoma. We're also working closely with the Ground Water Protection Council and the Interstate Oil and Gas Compact Commission to develop the next iteration of Frac Focus, the online chemical disclosure registry that is increasingly being used by states as a platform for public reporting of chemicals under their disclosure policies.

With that experience in mind, we're here today to speak to the aspects of SB315 that address transparency in industry operations – specifically, the so-called "spud to plug" approach to chemical disclosure.

First, we would like to commend the General Assembly and the Governor for the thoughtful approach that has been put forward. While the individual elements are similar to those required by several other states, Ohio deserves a great deal of credit for putting these standards together as a more complete package than other states have.

We think this shows exceptional foresight and recognition of the fact that, in order for Ohio to fully capture the benefits that come along with development of the resource, you have to have public acceptance that industry operations will be made safe for public health and the environment.

Transparency is a prerequisite to having fact-based conversations. It helps build public understanding of the relative risks associated with various oil and gas development activities. And it allows people to be partners instead of adversaries in solving problems.

Transparency also helps regulators and elected officials identify the strengths and weaknesses of regulatory structures, so scarce resources can be allocated more efficiently and effectively. Likewise, transparency helps regulators facilitate quick and effective responses to accidents and emergencies.

Finally, transparency helps companies learn from each other. This is a highly technical, data-driven industry. The greater the data availability, the greater the opportunity for companies to develop and operationalize practices to improve performance.

EDF has recommended a number of language changes on the bill in conversations with DNR staff and the Governor's office, and I'd like to express our thanks for their willingness to hear from us and for the receptiveness they've shown toward our suggestions. I would like to equally thank the Chair and members of the committee for the time they've given us leading up to these hearings. While we have additional suggestions for the bill, today I'm focusing our comments on three key areas that fall under the umbrella of transparency.

<u>Stimulation Fluid Chemical Disclosure</u>

EDF strongly supports the intent of the language for disclosure of stimulation fluid chemicals. We believe, however, that the bill language may have drafting ambiguities that could complicate the rule development process. We would welcome the opportunity to work with committee members on language changes to ensure the intent of the legislation is met and that

the final rules will include the following elements, consistent with the policies adopted in Colorado, Pennsylvania and other states, and which have been supported by both industry and environmental groups:

1. Disclosure of the type and volume of any base fluids.
2. Disclosure of all chemicals, not just those that are required to be listed on Material Safety Data Sheets.
3. Identification of chemical ingredients by both common name and CAS number.
4. Disclosure of chemical concentrations as a percentage of the total stimulation fluid (not as a percentage of the trade-name additives that contain the chemicals).
5. Disclosure of trade-name additives in stimulation fluids and descriptor of their general purpose (but not their concentrations).
6. Disclosure of chemical family names when chemical ingredient identities are claimed as trade secrets.
7. Provisions requiring a basic substantiation of the facts when trade secret claims are asserted.
8. Provisions ensuring DNR will have possession of any information for which trade secret claims are asserted so the agency can respond immediately to accidents or emergencies. These provisions should also make clear that the department will hold any such information confidential unless a trade secret claim is challenged and overturned.
9. Provisions that allow citizens to challenge trade secret claims under the Ohio Public Records Act.
10. Posting well-by-well disclosures on the DNR website and on user-friendly websites such as Frac Focus in a manner that will allow the public to search and sort data by chemical name, CAS number, operator, geographic area and time period.

<u>Disclosure of Chemicals Used in Drilling, Servicing, Operating and Plugging of Wells</u>

Unfortunately, with the onset of commercial development of unconventional oil and gas resources, the public debate on hydraulic fracturing chemicals has had the effect of obscuring the fact that production operations utilize a vast range of chemical products. Now, however, that is beginning to change, and the public is becoming aware that chemicals of concern are used throughout the lifecycle of a well.[1]

[1] As an example from just one phase of well development, please see attached below Appendix 8 from the International Petroleum Industry Environmental Conservation Association and International Association of Oil and Gas Producers report, "Drilling fluids and health risk management."

Ohio is to be commended for putting forward legislation to proactively address these concerns by requiring disclosure of the chemicals used throughout well development, production and abandonment. Doing so will help ensure citizens, oil and gas producers, regulators and elected leaders can have a rational, informed conversation about the potential risks associated with production operations.

Failing to follow through on the proposed bill language, in contrast, will almost certainly guarantee that Ohio will experience the acrimony and costs that come from public mistrust of industry. It can hardly be overemphasized that, when industry fights against transparency rules, the public naturally concludes it's because they're trying to hide something.

In addition to addressing public concerns, we would note that there is a very real, very immediate need for regulators to have this information.

Chemicals used in production operations can escape into the environment through a number of pathways, including: surface spills; exposure to subsurface formations and groundwater sources prior to casing and cementing (in the instance of drilling fluids); failures in well integrity and well control; failure to properly identify subsurface communication pathways, such as abandoned wells that pass through a target formation; failures in pits, impoundments and other containment facilities; improper waste handling and disposal; and volatilization into the air.

Regulators need to have a thorough understanding of what's being used in production operations in order to make a realistic determination about the immediate and longer-term risks and develop efficient and effective rules to mitigate those risks.

The language in SB315 will help ensure regulators have the information they need in order to adequately address the risks associated with various production operations and will help build public confidence that oil and gas operations will be conducted in ways that are protective of public health and the environment.

We note that similar requirements have been adopted in other oil and gas producing states. For example, Louisiana requires that documentation of all constituents added downhole in conjunction with drilling and workover operations [See LA Title 33, Part IX (708)(c)(3)(h)]. Colorado requires the chemicals used to treat injection water to be reported [See 316A COGCC Form 14]. The Bureau of Land Management requires reporting of the amounts and types for "Acid, Fracture, Treatment, Cement Squeeze, etc." on its well completion reports [See BLM Form 3160-4].

EDF would welcome the opportunity to work with the committee on modest language changes to ensure eventual agency rules meet the intent of the legislation and largely model the chemical disclosure methodologies that have been developed through cooperative efforts between environmental groups and industry, as outlined in the previous section of these comments.

Waste Characterization

SB315 proposes language to ensure injection well operators are aware of the chemicals used in drilling, stimulating, servicing, operating and plugging a well before accepting brine from that well. This is useful, but it does not account for the fact that produced water will contain contaminants beyond the chemicals that were sent down hole – including salts, metals, hydrocarbons, non-hydrocarbon organics and, potentially, naturally occurring radioactive materials – contaminants that may be picked up from the formation or that may be formed through interactions between oil field chemicals and formation materials.[2]

Moreover, SB315 only contemplates one disposal method – deep well injection. Whereas, produced water could ultimately go to reuse in subsequent wells, treatment and surface discharge, land application, road spreading and other forms of reuse. Likewise, the bill does not contemplate characterization, tracking and public reporting of wastes other than produced water – though other wastes such as drill cuttings, used drilling muds, and produced sands will carry a range of contaminants of concern.

Without a complete picture of the chemical makeup of produced water and other wastes, it is difficult to ascertain whether a particular method of treatment or disposition is protective of human health and the environment. The absence of such information may also handicap efforts to respond to and remediate accidental releases. And without such information, we forgo the benefits of public disclosure outlined at the top of these comments.

It is not our goal here to recommend particular protocols or conditions for characterizing, tracking and public reporting of oil field wastes. This is a complex topic that will require thoughtful, measured discussion between industry, legislators, regulators, environmental groups and the public to consider the questions of when operators should be required to characterize wastes, what types of analysis are appropriate for various situations, and what procedures need to be used for tracking and public reporting.

[2] Attached at the bottom of these comments are three tables from the 2011 Bureau of Reclamation report, "Oil and Gas Produced Water Management and Beneficial Use in the Western United States." These tables illustrate contaminants of potential concern in produced water.

Rather, our recommendation is that SB315 be amended to direct the DNR to gather stakeholder input, conduct a study and report to the General Assembly with recommendations for statutory and regulatory changes needed in order to implement requirements for the characterization of the chemical composition of wastes associated with production operations and for public reporting of that information.

We would also note that having moving toward a reasonable and useful policy on waste characterization would not place Ohio outside the norm. Pennsylvania, for example, requires operators to perform chemical analyses of residual wastes and report them annually to the state (See Pa. Code 287.54). Louisiana requires E&P waste characterization and reporting of disposal methods (See La. Code 43:XIX.503A). Colorado provides the Oil & Gas director discretion to require sampling and analysis of oil field wastes under certain circumstances; and as a matter of practice the Colorado Oil & Gas Conservation Commission reports requiring waste characterization under the authority provided by its 900 Series Rules (See 900 Series and COGCC response to STRONGER questionnaire at p. 6 of: cogcc.state.co.us/Library/ HydroFracStronger / COGCC_Response_To_STRONGER_06132011.pdf). Likewise, New York has in its proposed rules a requirement for operators to characterize and report wastes (See proposed 6 NYCRR Part 750-3.12).

Conclusion

Ohio is in an enviable position. It sits atop a tremendous energy resource that has the potential to bring much-needed economic development. It can even have an environmental upside if – and this is a big *if* – it's done right. Importantly, Ohio has the advantage of being able to learn from the successes and missteps of states that have already undergone intensive development of shale resources.

EDF is impressed with the thoughtfulness with which the General Assembly and the Kasich Administration have approached these issues – through measures put in place by SB165 in the 128th General Assembly, through a range of agency actions taken under the Governor's leadership and through the measures put forward in SB315.

These measures don't anticipate and won't solve every challenge that Ohio will face as development of the Utica shale intensifies, but they represent a very good start. EDF looks forward to working with members of this committee, leadership in the General Assembly and the Kasich Administration to improve and refine this legislation going forward.

Thank you again for the opportunity to appear on SB315. I would be happy to address any questions related to my testimony.

# *Appendix 8: Detailed health hazard information on drilling fluid components*

Information has been provided on generic chemical materials used in drilling fluid compositions; for specialty trade name products, reference must be made to the manufacturer's data for the specific product.

**Table 1: Health hazards of drilling fluid components**

| Component | Human health hazard |
|---|---|
| **Base fluid** | |
| Crude | Crude oil is raw petroleum extracted in its natural state from the ground and containing predominantly aliphatic, alicyclic and aromatic hydrocarbons. It may also contain small amounts of nitrogen, oxygen and sulphur compounds.<br><br>Crude oil is of low acute toxicity with dermal and oral LD50 values greater than 2000 mg/kg. Inhalation toxicity expected to be low. Light crude oils may pose an aspiration hazard and may also cause symptoms of central nervous system depression. Upon repeated exposure, some light crude oils may cause skin dryness or cracking. Available data indicate that crude oil is not a sensitizer. Data available indicate that crude oils are carcinogenic. |
| Diesel (gasoil) | Gasoils contain straight and branched chain alkanes (paraffins), cycloalkanes (naphthenes), aromatic hydrocarbons and mixed aromatic cycloalkanes (cycloalkanoaromatics). Most commercial gasoils contain polycyclic aromatic compounds (PAC's). In straight-run gasoil components these are mainly 2 and 3-ring compounds; with relatively low concentrations of 4 to 6-ring PAC's. The use of heavier atmospheric, vacuum or cracked gasoil components is likely to result in an increase in the content of 4 to 6-ring PAC's, some of which are known to be carcinogenic.<br><br>Skin exposure to diesel fuel will remove natural fat from the skin; repeated or prolonged exposure can result in drying and cracking, irritation and dermatitis. Excessive exposure under conditions of poor personal hygiene may lead to oil acne and folliculitis. A serious potential health hazard related to diesel fuel utilization concerns the possible risk of skin cancer under conditions of prolonged and repeated skin contact and poor personal hygiene. No epidemiological evidence exists for humans, but it has been demonstrated with mice that skin cancer can result in paint tests with light diesel oil and gasoils irrespective of the percentage of PAC's present. This effect is due to (chronic) irritation of the skin. The diesel fuels/gasoils that contain cracked components may also be genotoxic because of high proportions of 3-7 ring PAC's and their carcinogenicity may be much greater. Diesel fuels may contain 10% (w) or more PAC's. |
| Highly refined mineral oil | Highly refined mineral oils are of low acute toxicity and are not irritating or sensitizing. Data available indicate that highly refined mineral oils are not mutagenic, carcinogenic or reprotoxic. |
| Synthetic paraffin | Synthetic paraffins are of low acute toxicity. Data show that synthetic paraffins are not irritating or sensitizing. Based on their composition, synthetic paraffins are not expected to be mutagenic, carcinogenic or reprotoxic. |
| Linear alpha olefins | Based on screening level tests, alpha olefins are of low toxicity upon acute oral, dermal and inhalation exposure. Alpha olefins are slightly irritating to the skin and eyes of rabbits. In repeated dose studies alpha olefins of different chain length have shown comparable levels of low toxicity to female rats and male rat-specific kidney damage that is likely associated with the alpha2u-globulin protein. Based on screening level testing, alpha olefins appear not to be neurotoxic, produce no adverse effects on reproduction or foetal development, and are not genotoxic. As a result, all the above tested endpoints indicate a low hazard potential for human health. |
| Internal olefins | Olefins (alkenes) ranging in carbon number from C6 to C24, alpha (linear) and internal (linear and branched) demonstrate low acute toxicity by the oral, inhalation and dermal routes of exposure. Repeated-dose studies, using the inhalation (C6 alpha), dermal (C12-C16), or oral (C6 alpha and internal linear/branched; C8 and C14 alpha; and C16, C18 and C20-C24 internal linear/branched) routes of exposure, have shown comparable levels of low toxicity in rats. Based on evidence from neurotoxicity screens included in repeated dose studies, internal olefins are not neurotoxic. Based on evidence from reproductive/developmental toxicity screens in rats internal olefins are not expected to cause reproductive or developmental toxicity. Based on the weight of evidence alpha and internal olefins are not genotoxic. No carcinogenicity tests have been conducted on alpha or internal olefins; however, there are no structural alerts indicating a potential for carcinogenicity in humans. These materials are not eye irritants or skin sensitizers. Prolonged exposure of the skin for many hours may cause skin irritation. The weight of evidence indicates alpha and internal olefins with carbon numbers between C6 and C24 have a similar and low level of mammalian toxicity, and the toxicity profile is not affected by changes in the location of the double bond or the addition of branching to the structure. |

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**Table 1: Health hazards of drilling fluid components *(continued)***

| Component | Human health hazard |
|---|---|
| **Base fluid** | |
| Poly alpha olefin (PAO) | Poly alpha olefins are of low acute toxicity and are not irritating to eye and skin. |
| Esters | Data on C8-C16 fatty acid 2-ethyl hexanol ester indicate that this ester is of low oral acute toxicity. In an acute skin irritation test in rabbits, minimal skin irritation was observed. The ester is not primarily eye irritant.<br><br>The ester was non-genotoxic in a micronucleus test. Results from an oral repeated dose study in rats indicate that the ester is not toxic at up to 1000 mg/kg. |
| **Water** | |
| Fresh water | Fresh water is generally considered to be not hazardous to human health. |
| Sea water | Sea water has a low hazard potential for human health upon inhalation and dermal exposure. |
| Brine (see salts) | See Table 2 in Appendix 6, and below (osmotic—salts) |
| **Osmotic—salts** | |
| Calcium chloride ($CaCl_2$) | The acute oral and dermal toxicity of calcium chloride is low. The acute oral toxicity is attributed to the severe irritating property of the original substance or its high-concentration solutions to the gastrointestinal tract. In humans, however, acute oral toxicity is rare because large single doses induce nausea and vomiting. Irritation/corrosiveness studies indicate that calcium chloride is not/slightly irritating to skin but severely irritating to eyes of rabbits. Prolonged exposure and application of moistened material or concentrated solutions resulted in considerable skin irritation. The irritating effect of the substance was observed in human skin injuries caused by incidental contact with the substance or its high-concentration solutions. A limited oral repeated dose toxicity study shows no adverse effect of calcium chloride on rats fed on 1000–2000 mg/kg bw/day for 12 months. Calcium and chloride are both essential nutrients for humans and a daily intake of more than 1000 mg each of the ions is recommended. Genetic toxicity of calcium chloride was negative in the bacterial mutation tests and the mammalian chromosome aberration test. No reproductive toxicity study has been reported. A developmental toxicity study reveals no toxic effects on dams or fetuses at doses up to 189 mg/kg bw/day (mice), 176 mg/kg bw/day (rats) and 169 mg/kg bw/day (rabbits). (from: SIDS. *Screening Information Data Set for High Production Volume Chemicals*. (2005)). |
| Potassium chloride (KCl) | Potassium chloride is an essential constituent of the body for intracellular osmotic pressure and buffering, cell permeability, acid-base balance, muscle contraction and nerve function. Acute oral toxicity of KCl in mammals is low. In humans, acute oral toxicity is rare because large single doses induce nausea and vomiting, and because KCl is rapidly excreted in the absence of any pre-existing kidney damage. The toxicity upon repeated dose exposure is low. A threshold concentration for skin irritancy of 60 % was seen when KCl in aqueous solution was in contact with skin of human volunteers. The threshold concentration when applied to broken skin was 5%. No gene mutations were reported in bacterial tests, with and without metabolic activation. However, high concentrations of KCl showed positive results in a range of genotoxic screening assays using mammalian cells in culture. The action of KCl in culture seems to be an indirect effect associated with an increased osmotic pressure and concentration. No evidence of treatment -related carcinogenicity was observed in rats administered up to 1,820 mg KCl/kg body weight/day through the food in a two-year study. A developmental study revealed no foetotoxic or teratogenic effects of KCl in doses up to 235 mg/kg/day (mice) and 310 mg/kg/day (rats). Gastro-intestinal irritant effects in humans caused by KCl administrated orally have been reported at doses from about 31 mg/kg bw/day. One epidemiological investigation among potash miners disclosed no evidence of predisposition of underground miners to any of the diseases evaluated, including lung cancer. (from: SIDS. *Screening Information Data Set for High Production Volume Chemicals*. (2004)). |
| Sodium chloride (NaCl) | Sodium chloride is an essential nutrient for the normal functioning of the body. It is important for nerve conduction, muscle contraction, correct osmotic balance of extra cellular fluid and the absorption of other nutrients. Although rare, acute toxicity may be caused by ingestion of 500–1,000 mg sodium chloride/kg body weight. Symptoms include vomiting, ulceration of the gastrointestinal tract, muscle weakness and renal damage, leading to dehydration, metabolic acidosis and severe peripheral and central neural effects. Long-term effects of high (>6 g/day) dietary sodium chloride include the development of hypertension, and may increase the risk of kidney stone formation and left ventricular hypertrophy. In rodents, extremely high doses of sodium chloride during pregnancy caused musculoskeletal abnormalities, foetotoxicity and foetal death and post-implantation mortality and abortion. Sodium chloride has been demonstrated to be a gastric tumour promoter |

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**Table 1: Health hazards of drilling fluid components *(continued)***

| Component | Human health hazard |
|---|---|
| | In experimental animals and high sodium chloride intakes have been associated with incidence of stomach cancer in human populations with traditional diets of highly concentrated, salted foods. (Expert Group on Vitamins and Minerals (2003), www.food.gov.uk/science/ouradvisors/vitandmin/evmpapers) |
| Zinc bromide ($ZnBr_2$) | Zinc bromide inhalation can cause severe irritation of mucous membranes and upper respiratory tract. Symptoms may include burning sensation, coughing, wheezing, laryngitis, shortness of breath, headache, nausea and vomiting. High concentrations may cause lung damage. Ingestion of zinc bromide can cause severe burns of the mouth, throat, and stomach. Can cause sore throat, vomiting and diarrhoea. Ingestions are usually promptly rejected by vomiting, but sufficient absorption may occur to produce central nervous system, eye and brain effects. Symptoms may include skin rash, blurred vision and other eye effects, drowsiness, irritability, dizziness, mania, hallucinations, and coma.<br><br>Causes severe skin irritation with redness, itching and pain. May cause burns, especially if skin is wet or moist.<br><br>Can cause severe eye irritation or burns with eye damage.<br><br>Repeated or prolonged exposure by any route may cause skin rashes (bromaderma). Repeated ingestion of small amounts may cause central nervous system depression, including depression, ataxia, psychoses, memory loss, irritability and headache. |
| Calcium bromide ($CaBr_2$) | Calcium bromide brine is a highly concentrated aqueous solution of calcium bromide and calcium chloride. It is used extensively in the oil industry. This solution and its components are recognized as causes of skin injury and information is available from the manufacturers on their safe use and handling. Two patients who were injured following unprotected skin exposure to this solution and one patient who was injured following exposure to calcium chloride powder are reported. All sustained skin injuries characterized by an absence of pain and a delayed clinical appearance of the full extent of the injury. Furthermore healing was complicated by graft loss or was slow. Although organic bromine compounds are recognized as a cause of skin injuries, no previous reports of such injuries to humans secondary to calcium chloride or bromide exposure were found in the medical literature. Our experience with these patients is described. (Saeed *et al.*, Burns; 23 (7–8)[32]. 1997. 634–637) |
| Sodium bromide (NaBr) | Sodium bromide is of low acute oral toxicity. Sodium bromide is expected to be a slight to moderate eye irritant. Repeated or prolonged skin contact may cause irritation and superficial burns. Available data indicate that sodium bromide may act as a teratogen (behavioural effects). |
| Sodium formate (NaCOOH) | Sodium formate is of low acute oral toxicity (LD50 oral rat > 3000 mg/kg). Data show that sodium formate is slightly irritating to the eye. Based on read-across from caesium formate, sodium formate is expected to be slightly irritating to skin. Sodium formate is not expected to be a skin sensitizer. Sodium formate at 1% in drinking water did not produce clinically adverse effects in rats after administration for approximately 18 months. Sodium formate is not genotoxic *in vitro* or *in vivo*. Based on read-across with calcium formate, sodium formate is not expected to be reprotoxic or carcinogenic. |
| Potassium formate (KCOOH) | Potassium formate is of low acute oral toxicity (LD50 oral mouse 5500 mg/kg). Data show that potassium formate is slightly irritating to the eye. Based on read-across from caesium formate, potassium formate is expected to be slightly irritating to skin. Potassium formate is not expected to be a skin sensitizer. Repeated-dose toxicity tests are not available; however, the metabolite formic acid did not cause significant toxicity to rats when administered in their drinking water at 0.5 and 1% for 2 to 27 weeks. Based on read-across with calcium formate, potassium formate is not expected to be reprotoxic or carcinogenic. |
| Caesium formate (CsCOOH) | Caesium formate solution (83%) is harmful upon ingestion ($LD_{50}$ = 1780 mg/kg in rats), with clinical signs including depression, convulsions, respiratory distress, ataxia, and excessive salivation. Caesium formate monohydrate had low dermal (LD50 >2000 mg/kg) toxicity in rats, with signs of erythema noted at sites of application. Caesium formate solution (83%) was a slight skin irritant and a moderate eye irritant. Aqueous caesium formate (80% w/v) was not sensitizing to guinea pigs in a Buehler test. Repeated-dose toxicity tests are not available; however, the metabolite formic acid did not cause significant toxicity to rats when administered in their drinking water at 0.5 and 1 % for 2 to 27 weeks. The caesium cation is not expected to produce significant chronic toxicity. Caesium formate was not mutagenic in different *in vitro* assays. (National Industrial Chemicals Notification and Assessment Scheme (NICNAS), Australia, 2001). |

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**Table 1: Health hazards of drilling fluid components *(continued)***

| Component | Human health hazard |
|---|---|
| **Density (weighting agents)** | |
| Barite (Barium sulphate) | Studies in rats using a soluble salt (barium chloride) have indicated that the absorbed barium ions are distributed via the blood and deposited primarily in the skeleton. The principal route of elimination for barium following oral, inhalation, or intratracheal administration is in the faeces. Following introduction into the respiratory tract, the appearance of barium sulphate in the faeces represents mucociliary clearance from the lungs and subsequent ingestion. In humans, ingestion of high levels of soluble barium compounds may cause gastroenteritis (vomiting, diarrhoea, abdominal pain), hypopotassaemia, hypertension, cardiac arrhythmias, and skeletal muscle paralysis. Insoluble barium sulphate has been extensively used at large doses (450 g) as an oral radiocontrast medium, and no adverse systemic effects have been reported. No experimental data are available on barium sulphate; however, due to the limited absorption of barium sulphate from the gastrointestinal tract or skin, it is unlikely that any significant systemic effects would occur. The acute oral toxicity of barium compounds in experimental animals is slight to moderate. Barium nitrate caused mild skin irritation and severe eye irritation in rabbits. The lack of reports of skin or eye irritation in humans, despite its widespread use, suggests that barium sulphate, often used as a contrast medium, is not a strong irritant. Long-term studies of barium exposure in laboratory animals have not confirmed the blood pressure, cardiac, and skeletal muscle effects seen in humans and laboratory animals orally exposed to acutely high levels. Inhalation exposure of humans to insoluble forms of barium results in radiological findings of baritosis, without evidence of altered lung function and pathology. Animal studies involving respiratory tract instillation of barium sulphate have shown inflammatory responses and granuloma formation in the lungs; this would be expected with exposure to substantial amounts of any low-solubility dust, leading to a change in lung clearance and subsequently to lung effects. Currently available data indicate that barium does not appear to be a reproductive or developmental hazard. Barium was not carcinogenic in standard National Toxicology Program rodent bioassays. *In vitro* data indicate that barium compounds have no mutagenic potential. The critical end-points in humans for toxicity resulting from exposure to barium and barium compounds appear to be hypertension and renal function. The NOAEL in humans is 0.21 mg barium/kg body weight per day.<br>(www.inchem.org/documents/cicads/cicads/cicad33.htm, Concise International Chemical Assessment Document 33, Barium and barium compounds.) |
| Calcium carbonate | Acute effects may include irritation of skin, eyes, and mucous membranes. Based on an oral LD50 in rats of 6,450 mg/kg, calcium carbonate is of low oral acute toxicity. There is no adequate evidence for a tumour-promoting or genotoxic action of calcium carbonate. Effects on reproduction have not been shown. High dietary levels inducing maternal toxicity resulted in decreased fetal weights and delayed skeletal and dental calcification in rats and/or mice. There may be a silicosis risk in using impure limestone or chalk containing (3–20%) quartz. No adverse health effects have been reported in the literature among workers using calcium carbonate. High oral doses did not produce systemic toxicity in laboratory animals.<br>(Health Council of The Netherlands, 2003, calcium carbonate.) |
| Iron carbonate | Most data from iron compounds are derived from read-across with the ferrous salt iron sulphate. Ferrous sulphate has a low to moderate acute toxicity with a LD50 (rat) of 319–1,480 mg/kg. As ferrous sulphate is used in humans for the treament of anaemia, human data are also available. These indicate that the human LD50 is in the range of 40–1,600 mg/kg. Fatal doses are associated with gastric injury. Irritation data are scarce and indicate that ferrous sulphate may be irritating to skin and eyes. Ferrous sulphate is not a sensitizer. Genotoxicity data on ferrous sulphate are ambiguous. However. the overall weight of evidence from genotoxicity studies shows that ferrous sulphate is not genotoxic. Carcinogenicity is not expected. Ferrous sulphate is not a reprotoxicant. A study in calves fed up to 4000 ppm ferrous carbonate in diet shows that the calves were not affected. The tolerance for ferrous carbonate was higher than for ferrous sulphate. |
| Hematite | There are little data on hematite (or $Fe_2O_3$) available. The acute oral toxicity LD50 (rat) for $Fe_2O_3$ is greater than 10 g/kg. Upon eye contact some mechanical eye irritation may occur (dust). A carcinogenicity study in miners shows that haematite mining with low-grade exposure to radon daughters and silica dust was not associated with excess lung cancer in a relatively large cohort. |
| Ilmenite | There are no toxicity data available for ilmenite, or iron titanium oxide. It is expected that ilmenite is of low toxicity. There are no known hazards resulting from accidental ingestion of ilmenite sand as may occur during normal handling. Swallowing a large amount may result in irritation to the digestive system due to abrasiveness. Ilmenite dust may cause mechanical irritation of the eye. Ilmenite dust is regarded as general nuisance dust, but can be irritating if inhaled at high concentration. May cause symptoms such as coughing or sneezing. |

*continued ...*

**Table 1: Health hazards of drilling fluid components *(continued)***

| Component | Human health hazard |
|---|---|
| Manganese tetroxide | Manganese tetraoxide is of low acute toxicity. $Mn_3O_4$ is not a skin irritant, nor a sensitizer. $Mn_3O_4$ dust may cause some mechanical irritation to the eye. Results from a repeated dose study in monkeys and rats exposed to 11.6, 112.5 and 1,152 µ $Mn_3$ as $Mn_3O_4$ aerosol 24 h/day for 9 months, show no exposure related effects on pulmonary function, limb tremor or electromyographic activity.<br><br>There are several reports about manganese toxicity as a result of exposure to fume/vapour from elemental manganese and by inhalation of pyrolusite ($MnO_2$). Long-term inhalation (years) of manganese oxides may cause chronic manganese intoxication affecting the central nervous system, potentially leading to extensive disablement. Health risk of $MnO_2$ (widely described in literature) may be different from that of MnO, $Mn_2O_3$, and $Mn_3O_4$. The differences in oxidation states of the element Mn in these compounds may affect their bioavailability and distribution and thus their potential effects. |
| **Viscosity** | |
| Bentonite (or other clays) | An important determinant of the toxicity of bentonite and other clays is the content of quartz ($SiO_2$). Exposure to quartz is causally related to silicosis and lung cancer. Statistically significant increases in the incidence of or mortality from chronic bronchitis and pulmonary emphysema have been reported after exposure to quartz.<br><br>Single intratracheal injection into rodents of bentonite and montmorillonite with low content of quartz produced dose- and particle size-dependent cytotoxic effects, as well as transient local inflammation, the signs of which included oedema and, consequently, increased lung weight. Single intratracheal exposures of rats to bentonite produced storage foci in the lungs 3–12 months later. After intratracheal exposure of rats to bentonite with a high quartz content, fibrosis was also observed. Bentonite increased the susceptibility of mice to pulmonary infection. No adequate studies are available on the carcinogenicity of bentonite. Long-term occupational exposures to bentonite dust may cause structural and functional damage to the lungs. However, available data are inadequate to conclusively establish a dose-response relationship or even a cause-and-effect relationship due to limited information on period and intensity of exposure and to confounding factors, such as exposure to silica and tobacco smoke. (*Environmental Health Criteria*, Vol. 231 (2005) 159 p.) |
| Organophillic clay (montmorillonite, attapulgite, hectorite) | In general, the acute toxicity of organophillic clays is low. Some organophillic clays are used in cosmetics. Data on a hectorite clay show that the clay is of low acute and repeated dose toxicity. The clay is not a skin or eye irritant, nor a skin sensitizer. In dust form the hectorite clay may cause mechnical eye irritation. Data indicate that the hectorite clay is not genotoxic. Data on reprotoxicity and carcinogenicity are not available.<br>Check the MSDS for the compound-specific information. |
| Biopolymers | The biopolymers used are generally low toxicity compounds. Some of these biopolymers are also used as food additive. Check the MSDS for the compound-specific information. |
| Carboxymethyl cellulose | Carboxymethyl cellulose is of low toxicity. Carboxymethyl cellulose is approved for and used as food additive. (*WHO Food Additives Series*, Vol. 42 (1999) pp 175–9) |
| Polyanionic cellulose | Cellulose compounds are generally low toxicity compounds. Some of these cellulose compounds are also used as food additive. Check the MSDS for the compound-specific information. (*WHO Food Additives Series*, Vol. 40 (1998) pp 55–78; and *WHO Food Additives Series* Vol. 42 (1999) pp 175–9) |
| Guar gum (polysaccharide) | Guar gum is a low toxicity compound, commonly used as food additive. Allergic rhinitis following repeated inhalation exposure to guar gum dust has been reported. (Lagier *et al.*, 1990, *J. Allergy Clin Immun*, 85(4) p. 785–790; Kanerva *et al.*, 1988, *Clin Allergy*, 18(3) p245–252) |
| **Emulsifiers** | |
| Soaps | See Table 2. Several emulsifiers may irritate skin and/or eye and may be harmful by inhalation or if swallowed. Check the MSDS for the compound-specific information. |
| Amines | See Table 2. Several emulsifiers may irritate skin and/or eye and may be harmful by inhalation or if swallowed. Check the MSDS for the compound-specific information. |
| Imidazolines | See Table 2. Several emulsifiers may irritate skin and/or eye and may be harmful by inhalation or if swallowed. Check the MSDS for the compound-specific information. |
| Polyamides | See Table 2. Several emulsifiers may irritate skin and/or eye and may be harmful by inhalation or if swallowed. Check the MSDS for the compound-specific information. |

*continued ...*

**Table 1: Health hazards of drilling fluid components *(continued)***

| Component | Human health hazard |
|---|---|
| **Dispersants** | |
| Modified polyacrylates | Modified polyacrylates is a general term for several specific polyacrylates each having compound-specific toxicological properties. Check the MSDS for the compound-specific information. |
| Lignosulphonates | Lignosulphonates are complex polymers with a broad range of molecular mass and are derived from trees. The wood from trees is composed mainly of three components—cellulose, hemicellolose and lignin. In the sulphite pulping process, the lignins are sulphonated so they become water soluble and thus can be separated from the insoluble cellulose. A review of available toxicity data on several lignosulphonates by the US EPA indicated that lignosulphonates are of very low toxicity (www.epa.gov [Federal Register: February 16, 2005 (Volume 70, Number 31)]). The oral acute LD50 values are all greater than 2 g/kg. Repeated dose studies indicate NOAELs and LOAELs in the order of magnitude of g/kg/day. There is some (unsubstantiated) information that lignosulphonates given to rats before, during, and after mating at doses as high as 1,500 mg/kg/day did not cause adverse effects on reproduction or offspring. But at a dose level of 500 mg/kg/day there were histopathological changes in the lymph nodes of the mothers. There were no concerns identified for the mutagenicity or carcinogenicity of lignosulphonates. Based on the physical/chemical properties, and particularly on the large molecular weights of the lignosulphonates, lignosulphonates are not likely to be absorbed via any route of exposure. The only health effects of concern upon exposure to lignosulphonates are irritation of skin, eyes and respiratory system. For some lignosulphonates contact allergy has been reported (Andersson *et al.*, 1980; Contact Dermatitis 6(5): 354–355). The toxicity depends on the type and size of the lignosulphonate. Check the MSDS for the compound-specific information. |
| Tannins | Tannins are polyphenols derived from plants. Due to their ubiquitous presence in food they have been subject of many toxicity studies. In general, tannins are of low acute toxicity. Tannins in food have been associated with several beneficial and adverse health effects (Chung *et al.*, 1998 *Crit Rev Food Sci Nutr* 38(6): 421–64). IARC has evaluated tannic acid and tannins and concluded that although tannins were carcinogenic in animals upon subcutaneous injection, no epidemiological evidence in humans was available to evaluate their toxicity in humans. (*IARC Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Man: Some Naturally Occurring Substances*, Vol. 10, pages 253–262). Check the MSDS for the compound-specific information. |
| **Fluid loss** | |
| Synthetic polymers | Check the MSDS for the compound-specific information. |
| Carboxymethyl cellulose | See above (viscosity) |
| Polyanionic cellulose | See above (viscosity) |
| Starch | Starch is a low toxicity compound, commonly used as food additive. |
| Bentonite | See above (viscosity) |
| Modified lignites | Modified lignites are derived from brown coal. The toxicity mainly depends on the modification. Check the MSDS for the compound-specific information. |
| Asphalt | Asphalt, more commonly referred to as bitumen in Europe, is a dark brown to black, cement-like semisolid or solid or viscous liquid produced by the non-destructive distillation of crude oil during petroleum refining. When asphalts are heated, vapours are released; as these vapours cool, they condense. As such, these vapours are enriched in the more volatile components present in the asphalt and would be expected to be chemically and potentially toxicologically distinct from the parent material. Asphalt itself is considered to be of low toxicity. Asphalt fumes are the cloud of small particles created by condensation from the gaseous state after volatilization of asphalt. Symptoms associated with asphalt fume exposure are eye, nose, and throat irritation and coughing. These health effects appear to be mild in severity and transient in nature. Additional symptoms include skin irritation, pruritus, rashes, nausea, stomach pain, decreased appetite, headaches, and fatigue, as reported by workers involved in paving operations, insulation of cables, and the manufacture of fluorescent light fixtures. Asphalt fumes and vapours may be absorbed following inhalation and dermal exposure. Results of several *in vitro* mutagenicity studies on asphalt fumes are ambiguous. Results of carcinogenicity studies indicate that some asphalt fume condensates can cause tumours when applied dermally to mice. A meta-analysis of 20 epidemiological studies failed to find overall evidence for a lung cancer risk among pavers and highway maintenance workers exposed to asphalt. Under various performance specifications, it is likely that asphalt fumes contain carcinogenic substances.<br>(*Concise International Chemical Assessment Document* (CICAD) Vol. 59 (2004)) |

*continued ...*

**Table 1: Health hazards of drilling fluid components *(continued)***

| Component | Human health hazard |
|---|---|
| Resins | There are several types of resins, both naturally derived and synthetic. The toxicological properties vary. Some resins have been associated with skin irritation and allergic contact dermatitis. Check the MSDS for the compound-specific information. |
| Gilsonite | Gilsonite is a form of natural asphalt found in large amounts in the Uintah Basin of Utah. Workers can be exposed to the dust of gilsonite and to the fumes of gilsonite when heating or boiling the material. Gilsonite dust may cause mechanical eye irritation, while the fumes may be irritating to the eyes and respiratory system (Fairhall (1950) *Industrial Hygiene Newsletter*, 10(5): 9-10).<br><br>Industrial hygiene characterizations were performed by NIOSH at three gilsonite mills and nine gilsonite mining operations to measure occupational exposure to gilsonite and its constituents and to evaluate potential health effects (Kullman *et al., Am Ind Hyg Assoc J* (1989) 50(8): 413–418). Six out of seven bulk gilsonite samples had crystalline silica contents below 0.75% wt., no asbestos or other fibrous mineral compounds were detected in bulk samples from five different veins of gilsonite, and polynuclear aromatic hydrocarbons were not detected in any bulk samples. The authors conclude that the gilsonite dust exposure data are consistent with results of an earlier respiratory health survey of gilsonite workers in which the most definitive finding was an excess prevalence of bronchitic symptoms.<br><br>The respiratory health survey (Kelmig *et al., Am J Ind Med.* (1987); 11(3): 287–296) showed that increased prevalences of cough and phlegm were found in workers with high-exposure jobs, but no evidence for dust-related pulmonary function impairment was noted. |
| **Inhibition** | |
| Salts | See above (osmotic—salts) |
| Glycols (polyglycols) | Glycols, glycol ethers and polyglycols have different toxicities. Polyglycols are generally of low toxicity. Check the MSDS for the compound-specific information. |
| Silicate | The most commonly used silicates in drilling fluids are potassium silicate and sodium silicate. These ingredients combine metal cations (potassium or sodium) with silica to form inorganic salts. Sodium silicate administered orally acts as a mild alkali and is readily absorbed from the alimentary canal and excreted in the urine. The toxicity of silicates has been related to the molar ratio of $SiO_2/Na_2O$ and the concentration. Potassium and sodium silicates have a low to moderate acute toxicity. Rats orally administered 464 mg/kg of a 20% solution containing either 2.0 or 2.4 ratio to 1.0 ratio of sodium oxide showed no signs of toxicity, whereas doses of 1,000 and 2,150 mg/kg produced gasping, dypsnea, and acute depression. A case report describes that neutralized sodium silicate produced vomiting, diarrhea, and gastrointestinal bleeding in human.<br><br>Dermal irritation of potassium silicate and sodium silicate ranged from negligible to severe, depending on the species tested and the molar ratio and concentration tested. Potassium silicate was non-irritating in two acute eye irritation studies in rabbits. Sodium silicate was a severe eye irritant in acute eye irritation studies. A skin freshener (10% of a 40% aqueous solution) containing sodium silicate was non-irritating. Sodium silicate in another three eye irritation studies was highly irritating, irritating, and nonirritating, respectively. Detergents containing 7%,13%, and 6% sodium silicate mixed 50/50 with water were negligible skin irritants to intact and abraded human skin. A 10% of a 40% aqueous solution of sodium silicate was negative in a repeat-insult predictive patch test in humans. The same aqueous solution of sodium silicate was considered mild under normal use conditions in a study of cumulative irritant properties. Sodium silicate tested in elbow crease studies and semioccluded patch tests, produced low grade and transient irritation.<br><br>Repeated dose studies in Beagles and rats showed no overt signs of toxicity.<br><br>Sodium silicate was non-mutagenic in a standard bacterial assay. Reprotoxicity studies with sodium silicate in rats showed some effects on the number of offspring at high doses but no effects on male rat fertility.<br>(*Int J Toxicol* Vol. 24 Suppl. 1 (2005) pp 103–7) |
| Polyacrylamides (partially hydrolysed) | There are various different types of polyacrylamides. Check the MSDS for the compound-specific information. |
| **pH control** | |
| Sodium hydroxide (NaOH) | Sodium hydroxide is a skin and eye corrosive. In a human 4-hour patch test, sodium hydroxide (0.5%) was a very clear skin irritant. Irritation of the nose, throat, or eyes was observed in workers engaged in cleaning operations and in a small number of users of an oven spray. Ingestion might be fatal, as a result of, e.g., shock, infection of the corroded tissues, pulmonary necrosis, or asphyxia. No increase in mortality in relation to duration or intensity of exposure to caustic dust was found in a group of 265 workers for periods ranging from less than 1 year to up to 30 years. (Health Council of The Netherlands, 2000, sodium hydroxide) |

*continued ...*

**Table 1: Health hazards of drilling fluid components *(continued)***

| Component | Human health hazard |
|---|---|
| Potassium hydroxide (KOH) | Potassium hydroxide is a skin and eye corrosive. Following ingestion of (a solution of) potassium hydroxide, rapid corrosion and perforation of the oesophagus and stomach, stricture of the oesophagus, violent pain in throat and epigastrium, haematemesis, and collapse may occur. When inhaled in any form, potassium hydroxide is strongly irritating to the upper respiratory tract. Acute exposures may cause symptoms in the respiratory tract including severe coughing and pain. Additionally, lesions may develop along with burning of the mucous membranes. Inhalation may be fatal as a result of spasm, inflammation, and oedema of the larynx and bronchi, chemical pneumonitis, and pulmonary oedema (which can develop with a latency period of 5–72 hours). Chronic exposures may cause inflammatory and ulcerative changes in the mouth and possibly bronchial and gastrointestinal disorders. It has been reported that 10% of workers exposed to KOH during the production of ascorbic acid developed allergic dermatitis. At least one case of oesophageal carcinoma at the site of hydroxide-induced strictures has been reported. In mice, repeated applications of aqueous solutions (3-6%) of KOH to the skin for 46 weeks resulted in an increased incidence of skin tumours. Since tumourigenesis was associated with severe skin damage inducing marked epidermal hyperplasia, a non-genotoxic mechanism is assumed. (Health Council of The Netherlands, 2004, potassium hydroxide) |
| Calcium hydroxide ($Ca(OH)_2$) Lime | Acute exposures to calcium hydroxide may cause irritation, along with coughing, pain, and possibly burns of the mucous membranes with, in severe acute exposures, pulmonary oedema and hypotension with weak and rapid pulse. Solid calcium hydroxide is corrosive to the eyes and may cause severe injury to the skin. There are numerous case reports on accidental exposures to calcium hydroxide resulting in corneal and skin alkali burns and caustic ulcers. Generally, these effects are caused by the solid material and less commonly or rarely by solutions. Ingestion of alkali is reported to be followed by severe pain, vomiting (containing blood and desquamated mucosal lining), diarrhoea, and collapse.<br><br>Two epidemiological studies that addressed the association between cement-dust exposure and stomach cancer were considered insufficient to reach any conclusions on the association between cement dust exposure and stomach cancer. However, no adverse effects have been experienced by long-term exposed workers. Oral LD50 values of approximately 7,300 mg/kg bw were reported for rats and mice. No adequate repeated-dose toxicity (including carcinogenicity and reproduction toxicity) or genotoxicity/mutagenicity studies are available. (Health Council of The Netherlands, 2004, calcium hydroxide) |
| Citric acid | Based on many experimen tal data in animals and on human experience, citric acid is of low acute toxicity. The NOAEL for repeated dose toxicity for rats is 1200 mg/kg/d. The major, reversible (sub)chronic toxic effects seem to be limited to changes in blood chemistry and metal absorption/excretion kinetics. Citric acid is not suspected of being a carcinogen nor a reprotoxic or teratogenic agent. The NOAEL for reproductive toxicity for rats is 2500 mg/kg/d. Further, it is not mutagenic *in vitro* and *in vivo*. Also, the sensitizing potential is seen as low. In contrast, irritation, in particular of the eyes but also of the respiratory pathways and the skin, is the major toxicological hazard presented by citric acid. (SIDS. *Screening Information Data Set for High Production Volume Chemicals*, 2004) |
| Sodium bicarbonate ($NaHCO_3$) | Sodium bicarbonate is of low acute toxicity. Oral LD50 values are higher than 4,000 mg/kg bw, and an inhalation study in rats using a concentration of 4.74 mg/l inhalable dust produced no deaths. Sodium bicarbonate is slightly irritating to the skin and eye of rabbits. There is no indication of any adverse effects of long-term use or exposure via any route. *In vitro* bacterial and mammalian cell tests showed no evidence of genotoxic activity. Sodium bicarbonate is not a reprotoxicant. Based on the available information there are no indications that sodium bicarbonate has carcinogenic effects. Sodium bicarbonate has a long history of use in food and normal handling and use will not have any adverse effects. Acute oral ingestion of high doses may result in a ruptured stomach due to excessive gas development. Acute or chronic excessive oral ingestion may cause metabolic alkalosis, cyanosis and hypernatraemia. These conditions are usually reversible, and will not cause adverse effects. (SIDS. *Screening Information Data Set for High Production Volume Chemicals*, 2003) |
| Calcium oxide (CaO; Quick lime) | Occupational and accidental exposures have shown calcium oxide to be very irritating and corrosive to mucous membranes, eyes, and moist skin because of local liberation of heat and dehydration of tissues upon slaking of the small size particles and the resulting alkalinity of the slaked product (calcium hydroxide). Fatal burns have been reported after massive exposure. Calcium oxide was stated not to be sensitizing in an open epicutaneous test. Calcium oxide can cause severe irritation and burns to the eyes, oedema, hyperaemia, lachrymation, blurred vision, corneal opacities, ulceration, and perforation and loss of vision. Inflammation of the respiratory passages, ulceration, perforation of the nasal septum, and pneumonia have been attributed to inhalation of calcium oxide dust. Workers in lime factories for up to 40 years have experienced no ill effects from exposure to lime. (Health Council of The Netherlands, 2006, calcium oxide) |

*continued ...*

**Table 1: Health hazards of drilling fluid components *(continued)***

| Component | Human health hazard |
|---|---|
| **Wetting agent** | |
| Sulphonic acid | Sulphonic acids are a class of organic acids which have the tendency to bind to proteins and carbohydrates. The salts of sulphonic acids are the sulphonates. The toxicity of sulphonic acids depends on the specific type. Sulphonic acids may be irritating to skin and/or eye. Check the MSDS for the compound-specific information. |
| Amides | Amides are formed from the reaction of a carboxylic acid with an amine and are, compared to amines, very weak bases. The toxicity of amides depends on the specific type. Amides may be irritating to skin and/or eye. Check the MSDS for the compound-specific information. |
| Polyamides | There are different types of polyamides with a different toxicity. Check the MSDS for the compound-specific information. |
| **Rheological modifier** | |
| Fatty acids | Fatty acids are aliphatic monocarboxylic acids and can be naturally derived (from animal or vegetable fat) or synthetic (from oil or wax). Fatty acids are generally low toxicity compounds. Check the MSDS for the compound-specific information. |
| Polyacrylates | See above (dispersants) |
| **Filtration control** | |
| Asphalt | See above (fluid loss) |
| Lignite | See above (fluid loss) |
| Gilsonite | See above (fluid loss) |
| **Lubricating agents** | |
| Ester oils | Check the MSDS for the compound-specific information. |
| Asphalts | See above (fluid loss) |
| Graphite | In humans, the pathological and physiological response to inhaled graphite flake is similar to that induced by nuisance dusts and cause only transient pulmonary changes. Repeated exposure to very high concentrations may overwhelm the clearance mechanisms of the lung and result in pulmonary damage from the retained particles in unprotected individuals. However, these lesions either resolve with time or are of limited severity. Driver *et al.* (1993), *Govt Reports Announcements & Index* (GRA&I), Issue 06, 2094. |
| **Other** | |
| Bactericides | Check the MSDS for the compound-specific information. |
| Lost Circulation Material ($CaCO_3$, graphite, walnut shells, mica) | Lost circulation material may form a generic dust hazard. Mechanical irritation to the eyes and respiratory system may occur. |
| Ammonium bisulphate (max 63% aq. solution) | Ammonium bisulphate in solution is irritant to eyes and skin, and is irritating to the respiratory system. Inhalation of dust can produce irritation to gastro-intestinal or respiratory tract, characterized by burning, sneezing and coughing. |
| Sodium sulphite (approx 50% aq. solution) | Sulphites that enter mammals via ingestion, inhalation, or injection are metabolized by sulphite oxidase to sulphate. Sodium sulphite is of low to moderate acute toxicity. The oral mouse LD50 is 820 mg/kg. Exposure to the aerosol may irritate the upper respiratory tract. A three-day exposure of rats to a sodium sulphite aerosol produced mild pulmonary edema following exposure to 5 $mg/m^3$, and irritation of the tracheal epithelium with 15 $mg/m^3$. Between 2% and 5% of asthmatics are sulphite sensitive. Sodium sulphite may be irritating to skin and eyes. Positive reaction in human patch tests have been reported. Sodium sulphite is not considered to be reprotoxic; in rats, sodium sulphite heptahydrate at large doses (up to 3.3 g/kg) produced fetal toxicity but not teratogenicity. Sodium sulphite was negative in genotoxicity studies. IARC concluded that sodium sulphite is not classifiable (group 3) as to their carcinogenicity for humans. (*Int J Toxicol* Vol. 22, Suppl. 2 (2003) p. 63–88) |



Source: International Petroleum Industry Environmental Conservation Association and International Oil & Gas Producers report, "*Drilling fluids and health risks management: A guide for drilling personnel, managers and health professionals in the oil and gas industry*." OGP Report Number 396. 2009.

Table 14. Ranges of inorganic constituents in produced water[1]

| Constituent List | Units | Conventional | Unconventional |
|---|---|---|---|
| Antimony | mg/L | n/a | ND – 0.005 [e] |
| Aluminum | mg/L | < 0.50 – 410 [b,d] | 0.005 – 1.52 [f, g] |
| Arsenic | mg/L | 0.004 – 151 [a,b,d] | ND – 0.158 [e] |
| Barium | mg/L | ND – 1740 [a,b,d] | 0.445 – 125 [e, g] |
| Beryllium | mg/L | < 0.001 – 0.004 [c] | n/a |
| Bicarbonate | mg/L | ND – 14,750 [h] | 4.53 – 49,031 [g] |
| Boron | mg/L | ND – 95 [a,d] | 0.05 – 30.6 [e] |
| Bromide | mg/L | 150 – 1,149 [a,b] | ND – 41.1 [e] |
| Cadmium | mg/L | < 0.005 – 1.21 [a,b,d] | ND – 0.076 [e] |
| Calcium | mg/L | ND – 74,185 [h] | ND – 5,530 [e, g] |
| Chloride | mg/L | 2 – 254,923 [h] | ND – 52,364 [e, g] |
| Chromium | mg/L | ND – 1.1 [a,d] | ND – 3.71 [e, g] |
| Cobalt | mg/L | n/a | ND – 0.010 [e] |
| Copper | mg/L | < 0.002 – 5 [b,d] | 0.001 – 1.448 [e] |
| Fluoride | mg/L | n/a | 0.57 – 20 [f, g] |
| Iron | mg/L | ND – 1,100 [a] | 0.001 – 258 [e, g] |
| Lead | mg/L | 0.002 – 10.2 [b,d] | ND – 0.098 [e] |
| Lithium | mg/L | 3 – 235 [c,d] | ND – 1.50 [g] |
| Magnesium | mg/L | ND – 46,656 [h] | 1.2 – 918.9 [e] |
| Manganese | mg/L | < 0.004 – 175 [d] | ND – 3.11 [e, g] |
| Mercury | mg/L | < 0.001 – 0.002 [c] | ND – 0.014 [e] |
| Molybdenum | mg/L | n/a | ND – 0.448 [e] |
| Nickel | mg/L | < 0.08 – 9.2 [c] | ND – 0.082 [e] |
| Nitrogen, ammoniacal ($N\text{-}NH_3$) | mg/L | 10 – 300 [d] | n/a |
| Nitrate ($N\text{-}NO_3$) | mg/L | n/a | ND – 26.1 [g] |
| Potassium | mg/L | 0 – 14,840 [h] | ND – 1,100 [g] |
| Selenium | mg/L | n/a | ND – 1.27 [e] |
| Silver | mg/L | < 0.001 – 7 [c,d] | ND – 0.14 [g] |
| Sodium | mg/L | 1 – 149,836 [h] | 97.3 – 32,013 [e] |
| Strontium | mg/L | 0.02 – 6,200 [a,d] | ND – 47.9 [g] |
| Sulfate | mg/L | ND – 14,900 [h] | ND – 2,200 [e, g] |
| Tin | mg/L | ND – 1.1 [a] | n/a |
| Titanium | mg/L | < 0.01 – 0.7 [d] | n/a |
| Uranium | mg/L | n/a | ND – 2.5 [g] |
| Vanadium | mg/L | n/a | ND – 0.290 [e] |
| Zinc | mg/L | 0.01 – 35 [c] | 0.005 – 5.639 [e] |

[1] ND = nondetect; n/a = data not available; a = Fillo and Evans 1990; b = USEPA 2000; c = Shepard, Shore et al. 1992; d = Tibbetts, Buchanan et al. 1992; e = Cheung, Sanei et al. 2009; f = McBeth, Reddy et al. 2003; g = Colorado Oil and Gas Conservation Commission 2010; h = USGS 2002.

Table 15. Organic material in produced water from oil operations[1]

| Constituent | Units | Concentration Range | | | Technique (Method) |
|---|---|---|---|---|---|
| | | Low | High | Median | |
| TOC | mg/L | ND | 1,700 | NA | UV Oxidation/IR (USEPA 415.1) |
| COD | mg/L | 1,220 | | NA | Redox Titration (USEPA 410.3) |
| TSS | mg/L | 1.2 | 1,000 | NA | Gravimetric (USEPA 160.2) |
| Total Oil | mg/L | 2 | 565 | NA | Gravimetric (USEPA 413.1) |
| Volatiles | mg/L | 0.39 | 35 | NA | GC/MS (USEPA 1624 Rev B and USEPA 24 & CLP) |
| Total Polars | mg/L | 9.7 | 600 | NA | Florisil column/IR |
| Phenols | mg/L | 0.009 | 23 | NA | Silylation GLC/MS |
| Volatile Fatty Acids | mg/L | 2 | 4,900 | NA | Direct GLC/FID of water |

[1] ND = below detection limit; NA = not available.

Table 16. Volatile organics in produced water from gas operations[1]

| Constituent | Units | Concentration Range | | | Analytical Method | Reference |
|---|---|---|---|---|---|---|
| | | Low | High | Median | | |
| Benzene | mg/L | ND | 27 | NA | USEPA Method 1624 and 624 | Fillo, 1992 |
| Bis (2-chlorethyl) ether | mg/L | ND | 0.03 | NA | NA | GRI report, 1988 |
| Ethylbenzene | mg/L | ND | 19 | NA | USEPA Method 1624 and 624 | GRI report, 1988 |
| Phenol | mg/L | ND | 2.6 | NA | NA | GRI report, 1988 |
| Toluene | mg/L | ND | 37 | NA | USEPA Method 1624 and 624 | Fillo, 1992 |
| 2-Butanone | mg/L | ND | 0.37 | NA | NA | GRI report, 1988 |

[1] ND = below detection limit; NA = not available.

Source: U.S. Department of Interior Bureau of Reclamation, Reclamation: Managing Water in the West, Science and Technology Program Report No. 157, "*Oil and Gas Produced Water Management and Beneficial Use in the Western United States*," September 2011.