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**RE: An Analysis of Possible Increases in Exposure to Toxic Chemicals in Delta County, Colorado Water Resources as the Result of Gunnison Energy's Proposed Coal Bed Methane Extraction Activity**

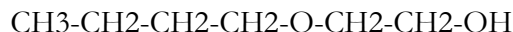
**BACKGROUND**

Gunnison Energy is proposing to extract coal bed methane in Delta County, Colorado. In its notices to the public it makes claims that "...the threats posed by hydraulic fracturing of CBM wells to USDWs [US drinking water supplies] are low and do not justify additional study." They also claim that the "...fluids used to extract coal bed methane from the ground do not substantially threaten public health."<sup>1</sup> The following addresses these claims and looks at possible direct and indirect health effects of CBM extraction on the citizens, domestic animals, and wildlife in Delta County.

**THE FRACTURING FLUIDS**

Gunnison Energy proposes to use a solvent, ethylene glycol monobutyl ether (2-butoxyethanol), hereafter designated as 2-BE, in a liquid fracturing mixture to facilitate the extraction of coal bed methane in Delta County. 2-BE will be present in the liquid component of the fluid at approximately 7 ppm (parts per million) based on data provided to Delta County Commissioners following three local Area Planning Committee meetings by Gunnison Energy Corporation (GEC), May 29, 2002.

The structural formula for 2-BE is:



2-BE is a highly soluble, colorless liquid with a very faint, ether-like odor.<sup>2</sup> At the concentration it is to be used in Delta County, it might not be detectable through odor or taste. 2-BE has low volatility, vaporizes slowly when mixed with water, and remains well dissolved throughout the water column.<sup>2</sup> Photolysis (degradation by sunlight) is not a factor in the breakdown of 2-BE. It mobilizes in soil and can easily leach into groundwater.<sup>2</sup> Because of these characteristics, it could remain entrapped underground for years and eventually migrate to a domestic well or to a surfacing spring. This contaminated water in

some cases might not reach wells, springs, and rivers in Delta County until long after GEC will have gone out of business.

The half-life of 2-BE in natural surface waters ranges from 7 to 28 days.<sup>2</sup> With an aerobic bio-degradation rate this slow, humans, wildlife and domestic animals could come into direct contact with 2-BE through ingestion, inhalation, dermal sorption, and the eye in its liquid or vapor form as the entrapped water reaches the surface. Aerobic biodegradation requires oxygen and therefore the deeper 2-BE is injected underground the longer it will persist. To date the aerobic biodegradation breakdown products of 2-BE have not been identified. The chemistry to detect the glycol ethers, including 2-BE, in environmental samples is very difficult and therefore there are few laboratories with the ability to accurately quantify its presence.<sup>2</sup>

## DIRECT HEALTH EFFECTS OF 2-BE

### **Immediate/Direct**

Following inhalation or swallowing, 2-BE is distributed rapidly to all tissues in the body via the blood stream in laboratory animals. When applied directly to the skin, 2-BE is rapidly absorbed.<sup>2</sup> In solution, it is absorbed more rapidly. It is broken down to its toxic component, 2-butoxyacetic acid (BAA) in both humans and laboratory animals following all three exposure pathways<sup>3</sup>. Breakdown and excretion of BAA through the urine is identical regardless of the pathway of exposure according to laboratory studies<sup>3</sup> No laboratory studies could be found that assessed cumulative effects from simultaneous ingestion, inhalation, and dermal exposure to 2-BE, which could be the scenario in Delta County.

### **Hemolytic Effects - Primary**

The most critical direct effect of 2-BE as the result of laboratory studies is its impact on red blood cells. It causes hemolysis (breakdown of red blood cells) by dissolving the fat in the cell membrane and causing the membrane to break down. 2-BE causes hematuria (blood in the urine) and blood in the feces. Blood appears in the urine as a result of kidney damage which can eventually lead to kidney failure. It is especially toxic to the spleen, the bones in the spinal column, and bone marrow (where new blood cells are formed) and the liver, where chemicals are detoxified (broken down for easy excretion from the body).<sup>2</sup> Chronic exposure can cause anemia, and in laboratory animals it leads to insufficient blood supply, cold extremities, and tail necrosis (a condition where the tail rots away).<sup>4</sup>

### **Other Effects - Secondary**

In a sub-chronic study over a period of 14 weeks, mice exposed to 2-BE exhibited the hemolytic effects mentioned above as well as a number of secondary problems involving the spleen and liver, and degeneration of kidney tubules.<sup>5</sup> In addition, females were more sensitive to fore-stomach necrosis, ulceration, and inflammation occurring at half the dose required to cause the same problems in males. Female fertility was also significantly reduced in mice because of embryo mortality.<sup>6</sup> In this study, the dead embryos were discarded, and as a result, the prenatal effects of 2-BE on the embryos were not determined.

EPA recommends that 2-BE be classified as a mild eye irritant.<sup>3</sup> However, a recent study published after EPA reached this classification could lead to a higher risk classification. Using oral exposure in rats, severe damage to the eye was discovered that led to retinal

detachment, photoreceptor degeneration and occlusion resulting from multiple thrombosis of the blood vessels in the eye.<sup>7</sup> In this study, females were more susceptible.

With few exceptions most of the evidence mentioned above was derived from inhalation studies. All of the studies used standard, high-dose testing protocols to detect obvious birth defects and organ damage, cancer, mutations, convulsions, and skin and eye irritation. No long-term, multigenerational, chronic oral studies at environmentally relevant concentrations are available that could rule out prenatal damage.

### **Immunotoxicity**

Early studies suggested that perhaps 2-BE does not affect the immune system<sup>8,9</sup> more recent studies using more sophisticated measures and lower doses have determined otherwise. In an early immunotoxicity study, the lowest doses significantly increased the natural killer (NK) cell response in males and females, and the highest doses induced no response.<sup>9</sup> The investigators never did find the lowest dose at which there would be no effect. However, they did not consider this an indication of adversity.

In another study, rats exposed to 2-BE in water for 21 days showed no structural effects in the liver or the testes, however their livers were significantly heavier and the animals experienced reduced body weight even at the lowest dose. However, they were surprised to find that at the lowest 2-BE dose NK cell responses were increased. A more recent study exposing female mice topically for 4 days once again confirmed the elevated NK cell response.<sup>10</sup>

A 2002 study reports that 2-BE at unusually low doses inhibits a normal contact hypersensitivity response in female mice.<sup>11</sup>

### **Carcinogenicity**

At the end of a two year chronic bioassay, elevated numbers of combined malignant and non-malignant tumors of the adrenal gland were reported in female rats and male and female mice.<sup>5</sup> Low survival rates in the male mice in this study may have been the result of the high rate of liver cancers in the exposed animals.<sup>5</sup> This study revealed that long-term exposure to 2-BE often led to liver toxicity before the hemolytic effects were discernible.<sup>5</sup>

No human epidemiological studies are available to assess the potential carcinogenicity of 2-BE. However, from the results of laboratory studies, using Guidelines for Carcinogenic Risk Assessment (1986), 2-BE has been classified by the USEPA as a *possible human carcinogen*.<sup>3</sup>

### **SENSITIVE POPULATIONS**

A number of laboratory studies confirmed that aging increases susceptibility to the effects of 2-BE. Older animals have reduced ability to metabolize the toxic metabolite BAA and this, combined with reduced kidney function that accompanies aging reduces their ability to excrete it in the urine.<sup>3</sup>

Females are more susceptible to the hematological effects in laboratory animal and human studies. There is an obvious gender and age sensitivity to 2-BE in humans as determined from accidental poisonings with females being more sensitive. In addition, among humans there may be sub-populations that might be more sensitive than others.<sup>3</sup>

A list of risk factors for people exposed to 2-BE includes those:

- (1) using the pharmaceuticals hydralazine, dilantin, chloramphenicol, and sulfonamides;
- (2) with infections, such as herpes, malaria, parasites, and rubella;
- (3) with a family history of gallstones, cholecystectomy, jaundice, Rh and APO positive;
- (4) with iron deficiency; and
- (5) with systemic illnesses, such as cardiac, gastrointestinal, liver, and kidney disease, and hypothyroidism.<sup>3,12</sup>

From a wildlife and domestic animal perspective, it is important to note that a variety of studies with laboratory animals revealed that some species are more sensitive to 2-BE than others.<sup>3</sup> For example, rats are more sensitive than mice to the toxic effects of 2-BE on the liver. No studies were found using wildlife or domestic animals.

#### INDIRECT HEALTH EFFECTS OF 2-BE

2-BE is widely used as an emulsifying agent and as a solvent for mineral oils<sup>2</sup>. This makes it an excellent candidate for releasing the natural, oily, coal-tar hydrocarbons found in coal that have been recognized for over a century to cause cancer.

#### CUMULATIVE AND AGGREGATE HEALTH HAZARDS

As mentioned above, no cumulative exposure studies have been done that evaluate the simultaneous impact of ingestion, inhalation, and topical exposure to 2-BE, which could be the mode of exposure to residents in Delta County. If 2-BE comes directly into the home via a well it will be used for drinking, bathing, showering, and doing laundry and dishes. Laboratory studies have revealed that in the case of bathing or applying 2-BE to the skin, it is readily absorbed through the skin rather than volatilizing. If water containing 2-BE is heated, as it comes out of the tap some of the 2-BE will off-gas into the home environment. Most of the studies mentioned above used inhalation as the pathway of exposure to 2-BE. Inhalation of 2-BE in the home could become a problem. For example, concern about exposure to the volatile by-products (trihalomethanes or THMs) in chlorine treated tap water<sup>13</sup> led to the discovery that taking a bath or a shower can lead to excessively high dose exposure to THMs. This exposure can exceed the level of exposure from drinking the water and add to the dose from drinking the water. Because of the volatility of 2-BE, the same pathway of exposure could become of concern for Delta County residents if 2-BE reaches their wells and especially if the water is heated.

Of increasing concern by federal health agencies are the *unpredictable*, interactive effects of mixtures of chemicals.<sup>14</sup> Under the scenario described in Gunnison Energy's prospectus, the concentrations of three classes of chemicals that are toxic individually at very low concentrations could become introduced or increased in the environment of Delta County. These include (1) the trace elements arsenic, molybdenum, and selenium, already a problem in Delta county, (2) a synthetic solvent, 2-BE, and (3) the polyaromatic hydrocarbons and coal tars found in coal beds. Arsenic, 2-BE, and aromatic coal bed tar derivatives are known carcinogens. In aggregate, whether their effects would be additive or synergistic has not been determined. However, in one study, the authors were surprised to find that 2-BE potentiated the lethality of low level exposure to another toxicant, a bacterially produced lipopolysaccharide (LPS) that is found in the human gut under certain conditions.<sup>8</sup>

Additional contamination of potable water could come from the impurities in the 2-BE product used in the extraction process. Commercial grade 2-BE can range in impurities depending upon the production process, manufacturer, and grade of the solvent. One impurity, sodium hydroxide (lye), a strong caustic, might possibly contribute to the alkalinity of the water. It was discovered in one product at 0.25%. Even high grade 2-BE with greater than 99% purity can contain 0.2% w/w ethylene glycol (anti-freeze), diethylene glycol, and diethyl monobutyl ether, sister compounds to 2-BE with much higher toxicity.<sup>2</sup>

## ENVIRONMENTAL EFFECTS

### **Increased salinity**

2-BE leaves an alkaline residue upon evaporation which might slightly add to the alkalinity problem that increases as surface water approaches the lower reaches of Delta County. Because of the solubility of sodium salts they can travel long distances in rivers and could increase the salinity problem in the Colorado River downstream.

Locally, any additional water that increases the salinity could also increase the mobilization of some of the alkaline soluble, problem elements such as arsenic and selenium, already posing health risks in Delta County. Health advisories are already in effect for Sweitzer Lake warning people not to eat the fish because of the high levels of selenium in the fish tissue.

A peer reviewed report by the US Forest Service on the threat of increased selenium contamination in the Mancos and La Plata River drainages describes a scenario similar to the Gunnison River drainage in Delta County where selenium is already at levels of concern.<sup>15</sup> The hazards include threats to wetlands, aquatic habitat, invertebrates, fish, birds and other wildlife reproduction. Delta County is in a unique and fragile situation – (1) it already has the natural geological existence of selenium, (2) its local hydrology that has been embellished and complicated through extensive irrigation activity, and (3) a climate prone to drought .

There is a growing collection of scientific papers on the adverse health effects of selenium in wildlife exposed to elevated concentrations of selenium in seep-like situations (natural and human-induced) in the West. Waterfowl, fish, and invertebrates have experienced decreased hatching success and increased birth defects as a result of exposure in the egg. Chicks of avocets, stilts, ducks, coots, etc. have been found with crossed bills, missing eyes, and other deformities in aquatic systems where irrigation run off water collects.

## HEALTH RISKS TO BE TAKEN INTO CONSIDERATION

Although no standard has been established yet for 2-BE in drinking water, in 1993 the EPA set a minimum risk level (MRL) for 2-BE at 0.07 mg/kg/day based on an adult 70 kg male drinking two liters of water a day. This value is based on liver toxicity studies in rats and not on more sensitive immune, developmental, and functional health effects that have become of concern over the past decade. In 1998 EPA derived a reference dose RfD for 2-BE at 0.5 mg/kg/day for non-cancer effects. This is based on lifetime exposure. EPA admits “ Since drinking water exposures are highly complex and variable, a simplifying assumption was used in all simulations ....”. EPA had no human data to derive its value.<sup>3</sup>

GEC is planning to inject fluid into the ground in Delta County at 7 ppm. If this fluid reaches the taps in Delta County at that concentration, it will be providing 0.2 mg/kg/day

per two liters of water, approximately three times higher than the MRL and a little more than half the RfD.

#### RECOMMENDATIONS

1. First and most important, it is imperative to understand the hydrology of Delta County better. In addition, the complex diversions of potable water for irrigation and domestic use throughout the county must be factored into this knowledge.
2. Second, it is imperative to determine the current concentrations of the toxic chemicals in the coal bed water to be released during extraction prior to introducing the fracturing liquids. This must include the entire scope of trace elements from alkaline to acid based derivatives in both their dissolved and suspended form. In addition, the entire scope of polyaromatic hydrocarbons (both parent and alkylated forms) in the underground coal bed water should be quantified prior to any activity. Because of the toxicity of the elements and compounds of concern, detection limits throughout this monitoring should be no higher than a part per trillion. Information such as this will allow for determining if the fracturing liquid releases additional toxic components, and in the case of the PAHs, through dissolution by the 2-BE.
3. Throughout the mining life of the well, the underground fluid with which it will interface should be monitored on a regular basis for its toxic components. See those components mentioned in Number 2. If the concentrations of the contaminants decrease, this could indicate that precious potable subsurface or surface water is being drained from above. This provides an approach for detecting dewatering before too much potable water is lost.
4. If exploration begins, GEC must keep daily inventories of the total amount of fracturing liquid injected, including the exact amount of each component in the fluid.
5. GEC should be required to retrieve all surfacing liquid for containment. The volume of the retrieved liquid should be reported and the concentrations of the chemicals in that liquid quantified on a regular basis for auditing purposes to account for the toxic chemicals that were introduced under Number 4.
5. GEC's plans for disposal of this toxic liquid should be presented to the residents of Delta County for approval before any leases are approved.
6. Any changes in the composition of the fracturing liquid must be reported to the citizens of Delta County for consideration before the liquid is used.
7. If GEC should find that it needs or wants to use anything other than sand for propping, it must provide to the citizens of Delta County for consideration all the components in the alternative material before the material is used. The purity of the alternative products used must be provided as well. Trade names will not be acceptable.

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<sup>1</sup> The Daily Sentinel, Sunday, September 8, 2002. p. 8C

<sup>2</sup> Agency for Toxic Substances and Disease Registry . US Department of Health and Human Services. (1998) Toxicological Profile of 2-Butoxyethanol and 2-Butoxyethanol Acetate.

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<sup>3</sup> US Environmental Protection Agency. Toxicological Review of Ethylene Glycol Monobutyl Ether (EGBE) In Support of Summary Information on the Integrated Risk Information System (IRIS), October 1999

<sup>4</sup> Nyska A, Maronpot RR, PH Long, JH Roycroft, JR Hailey, GS Traylor, BI Ghanayem (1999) Disseminated thrombosis and bone infarction in female rats following inhalation exposure to 2-butoxyethanol. *Toxicol Pathol* 27(3):287-294.

<sup>5</sup> National Toxicology Program (NTP). 1998 NTP Technical report on the toxicology and carcinogenesis studies of 2-butoxyethanol (Cas No. 111-76-2) in F344/N rats and B6C3F1 mice (inhalation studies). US Department of Health and Human Services, Public Health Service, National Institutes of Health, Research Triangle Park, NC NTP TR 484. NIH Draft Publ. No. 98 -3974.

<sup>6</sup> Heindel, JJ, Gulati, DK, Russell, VS, et al. (1990) assessment of ethylene glycol monobutyl and monoethyl ether reproductive toxicity using a continuous breeding protocol in Swiss CD-1 mice. *Fundam Apply Toxicol* 15:683-696.

<sup>7</sup> Nyska A, RR Maronpot, BI Ghanayam. (1999) Ocular thrombosis and retinal degeneration induced in female F344 rats by 2-butoxyethanol. *Hum Exp. Toxicol* 18(9):577-582.

<sup>8</sup> Smialowicz, RJ, Williams, WC, Riddle, MM. et al. (1992). Comparative immunosuppression of various glycol ethers orally administered to Fischer 344 rats. *Fundam Apply Toxicol* 18:621-627.

<sup>9</sup> Exon JH, GG Mather, JLBussiere, DP Olson, PA Talcott. (1991) Effects of subchronic exposure of rats to 2-methoxyethanol or 2-butoxyethanol: thymic atrophy and immunotoxicity. *Fundam Appl Toxicol* 16(4):830-840.

<sup>10</sup> Singh P, Zhao S, Blaylock RL. (2001). Topical exposure to 2-butoxyethanol alters immune responses in female BALB/c mice. *Int Jrl Toxicol* 20:383-390.

<sup>11</sup> Singh P, Morris B, Zhao S, Blaylock RL. (2002) Suppression of the contact hypersensitivity response following topical exposure to 2-butoxyethanol in female BALB/c mice. *Int Jrl Toxicol*, 21:107-115.

<sup>12</sup> (Berliner N, Duffy, TP, Abelson HT. (1999) Approach to adult and child anemia. In: Hoffman, R ed. *Hematology: Basic Principles and Practice*. 2<sup>nd</sup> ed. New York, NY: Churchill Livingstone, pp.468-483.

<sup>13</sup> Nester AM, Singer PC, Ashley DL, Lynberg MC, Mendola P, Langlois PH, Nichols JR. (2002). Comparison of trihalomethanes in tap water and blood. *Env Sc Techn*. 36(8):1692-1698.

<sup>14</sup> Department of Health and Human Services, Agency for Toxic Substances and Disease Registry, (2001). *Guidance Manual for the Assessment of Joint Toxic Action of Chemical Mixtures*. Draft for Public Comment.

<sup>15</sup> Lemly AD (1997). Environmental hazard of selenium in the Animas La Plata water development project. *Ecotoxicol Environ Safety* 37:92-96.