

STATE OF RHODE ISLAND AND PROVIDENCE PLANTATIONS  
ENERGY FACILITY SITING BOARD

IN RE: INVENERGY THERMAL DEVELOPMENT LLC's :  
APPLICATION TO CONSTRUCT THE : DOCKET NO. SB-2015-06  
CLEAR RIVER ENERGY CENTER IN :  
BURRILLVILLE, RHODE ISLAND :

**DIRECT TESTIMONY OF ERIC EPNER, P.E.,**  
**ON BEHALF OF THE TOWN OF BURRILLVILLE**

**SUMMARY OF ERIC EPNER**  
**DIRECT TESTIMONY**  
**PROPOSED CREC, TOWN OF BURRILLVILLE, RI**

Eric Epner, PE, Vice President and Department Manager of Fuss & O'Neill, Inc., is an air emission compliance expert and he is testifying that, based on the information submitted to DEM to date, the disadvantages to air quality of the proposed Invenergy facility within the Town of Burrillville outweigh any potential benefits.

Mr. Epner provides an evaluation of the Major Source Air Permit Application (and addendum) for a combined-cycle electric generating facility, the Air Dispersion Modeling Report, the Multisource Modeling Addendum, the Health Risk Assessment Report, the Revised Water Supply Plan, and related documents, including data responses and report amendments and updates. He identified inconsistencies/flaws noted in the Major Source Permit Application (and addendum), the Air Dispersion Modeling Report, the Multisource Modeling Addendum, and the Health Risk Report. Further, he comments upon the disadvantages to the local air quality if the Invenergy facility were to be sited within Burrillville (e.g., emission reduction credits, vehicular emissions from trucks, decline in air quality from facility emissions, etc.).

1 **Q. Please state your name and business address.**

2 A. My name is Eric Epner. My Rhode Island office is located at 317 Iron Horse Way, Suite  
3 204, Providence, Rhode Island 02908.

4  
5 **Q. By whom are you employed and in what capacity?**

6 A. I am employed by Fuss & O'Neill, Inc., as a Vice President and Department Manager. I lead  
7 our Facilities Compliance Department. Fuss & O'Neill is a multi-disciplinary engineering  
8 firm that specializes in environmental compliance and environmental permitting.

9  
10 **QUALIFICATIONS**

11 **Q. Please describe your educational background and your professional experience.**

12 A. I received my Bachelor of Science (BS) in Chemical Engineering from Clarkson University  
13 located in Potsdam, New York in 1985. I am a Professional Engineer (PE) registered in the  
14 Commonwealth of Massachusetts, the State of Connecticut, the Commonwealth of Virginia,  
15 and the State of South Carolina. I have over 31 years of experience in environmental  
16 compliance and permitting. My resume was filed with this Board on September 9, 2016.

17  
18 **Q: What are your technical specialties?**

19 A. My experience spans a wide variety of environmental engineering projects including air,  
20 environmental, health, and safety compliance auditing, water, and solid/hazardous waste  
21 permitting, storm water compliance, petroleum storage and handling compliance, and  
22 remediation. My projects have spanned many client sectors and geographies. My principal  
23 focus areas are air permitting and air pollution control.

24

1 **Q. Have you ever testified as an expert witness before any court or administrative body?**

2 **If so, what was the nature of your testimony?**

3 A. I have previously testified at numerous public hearings for the environmental permitting of  
4 industrial facilities. Generally, my testimony has focused upon a facility's environmental  
5 impacts and/or its compliance with environmental regulations.

6  
7 **PURPOSE**

8 **Q. What is the purpose of your testimony in this case?**

9 A. The purpose of my testimony is to review the air emission impacts of Invenergy's proposed  
10 electrical generating project located in Burrillville. This project is known as the Clear River  
11 Energy Center (CREC). I provided an independent third party review to the Town of  
12 Burrillville. I advised the Town on deficiencies in Invenergy's submitted plans and permit  
13 applications.

14  
15 **Q. What materials have you reviewed in this matter?**

16 A. I have reviewed the following key documents:

- 17 • Air Dispersion Modeling Protocol, CREC, prepared by EES Group, dated April 20,  
18 2015;
- 19 • Major Source Permit Application, CREC, prepared by ESS Group, dated June 26, 2015;
- 20 • Health Risk Assessment Protocol, CREC, prepared by ESS Group, dated June 26, 2015;
- 21 • Rhode Island Department of Environmental Management (RIDEM) comments related  
22 to the Health Risk Assessment Protocol, RIDEM, August 11, 2015;
- 23 • RIEFSB Application, CREC, prepared by ESS Group, dated October 28, 2015;

- 1 • Air Dispersion Modeling Report, CREC, prepared by ESS Group, dated October 30,
- 2 2015;
- 3 • Health Risk Assessment Report, CREC, prepared by ESS Group, dated January 27,
- 4 2016;
- 5 • Major Source Permit Application Addendum, CREC, prepared by ESS Group, dated
- 6 September 15, 2016;
- 7 • Multisource Modeling Addendum, CREC, prepared by ESS Group, dated October 18,
- 8 2016;
- 9 • Revised Water Supply Plan, CREC, prepared by ESS Group, dated January 11, 2017;
- 10 • Invenergy's responses data requests from the Town;
- 11 • Related filings, correspondence, and other documents concerning the CREC.

12

13 **AIR PERMIT APPLICATION**

14 **Q. Please summarize your evaluation of the deficiencies of the Air Permit Application**

15 **for the proposed CREC.**

16 A. The primary focus of my evaluation has been my review of the Major Source Air Permit

17 Application for a Combined-Cycle Electric Generating Facility and subsequent Addendum

18 ("Air Permit Application"), the Air Dispersion Modeling Report and subsequent Multisource

19 Modelling Addendum ("Modelling Report"), and the Health Risk Assessment Report. The

20 Air Permit Application is a permitting document for a facility which has the potential to emit

21 air pollutants above a given threshold (the threshold varies by pollutant). The Application

22 for those facilities that have significant air emissions is submitted to and approved by

23 RIDEM. It includes information on the sources, amounts, and effects of estimated or

24 modeled air pollutants generated by a proposed facility.

1  
2 The Health Risk Assessment Report is a report, also submitted to RIDEM, which evaluates  
3 the risks and potential impacts of a proposed facility to human health. The evaluated risk  
4 includes the likelihood that a given exposure or series of exposures will damage the health of  
5 individuals. The assessment is the process to estimate the nature and probability of adverse  
6 health effects in humans who may be exposed to air contaminants, now or in the future.

7  
8 Each of the noted documents prior to the issued Addenda had significant inconsistencies,  
9 omissions, and errors. Some of the inconsistencies noted in the original Major Source Permit  
10 Application submitted to RIDEM included:

- 11 • the Property Line and Fence Line locations;
- 12 • Inconsistencies in statements regarding the proposed use of Ultra-Low Sulfur Diesel  
13 (ULSD) as a secondary fuel in the Combustion Turbines;
- 14 • Proposed air permit limits for use of ULSD in the Combustion Turbines;
- 15 • ULSD storage tanks (size, number, location);
- 16 • General emission calculations;
- 17 • The Best Available Control Technology/Lowest Achievable Emission Rate  
18 (BACT/LAER) selection consistent with the methodology described in the report text.

19 These were not the only errors, omissions or inconsistencies.

20  
21 Many of the errors and omissions were addressed in the subsequent Air Permit Application  
22 Addendum and the Multisource Modelling Addendum. However, all issues and questions  
23 were not fully addressed. Moreover, the Addenda in some instances raised new concerns  
24 and questions. These outstanding issues are outlined below for each specific document.

1 Additionally, the revised Table 1, Facility Potential Emission Summary, contains an  
2 additional column for Gas Turbines/HRSG/Duct Burner Steady State Operations,  
3 identified as “N.Gas w/DB”. This may represent natural gas firing with duct burners.  
4 However, it is not clear if this scenario was not considered in previous permit application  
5 materials, or if this was the previous application assumption. Additional clarification is  
6 needed to distinguish this from other “N.Gas” column, to determine what scenario was  
7 previously considered in 2015 application, and what facility information triggered this  
8 additional column/information.

#### 10 AIR DISPERSION MODELING REPORT

11 **Q. Please summarize the deficiencies in the Air Dispersion Modeling Report, and**  
12 **subsequent Multisource Modelling Addenda.**

13 A. Looking at the Air Dispersion Modeling Report in its original form submitted to RIDEM,  
14 inconsistencies in this report included:

- 15 • Flawed emission calculations and input parameters taken from the Major Source Permit  
16 Application noted above;
- 17 • AERMOD (modeling software) emission source locations;
- 18 • Errors/inconsistencies in equipment specifications and operating parameters;
- 19 • Errors on Figures included in the Report (Figures, 3, 6, 8);
- 20 • AERMOD receptors;
- 21 • Missing and/or inconsistent information contained within the Tables (Tables 4, 5, 16).

22 Many of the noted inconsistencies were addressed in the subsequent Addendum. However,  
23 there remains one large issue. The revised Multisource Modelling Addendum relies wholly  
24 on conditions described in a Minor Source Air Permit Modification Application submitted

1 for the adjacent Algonquin Compressor Station, which is owned and operated by Spectra  
2 Energy. This Application has been submitted by Spectra to RIDEM for review. However,  
3 prior to issuance of a modified permit for Spectra, there is no contractual duty for the  
4 Algonquin Compressor Station to adhere to the terms of the permit application for this  
5 wholly unrelated adjacent facility. At any time, Spectra Energy could rescind the application  
6 materials and continue operations as is. Conversely, RIDEM could choose not to approve  
7 the permit application submitted by Spectra. In either of these situations, the input  
8 parameters of the Multisource Modelling Addendum would be incorrect. Therefore, I am of  
9 the opinion that it would be improper for the EFSB to rely on the modeling results unless  
10 and until the Spectra permit is issued.

### 11 HEALTH RISK ASSESSMENT REPORT

12 **Q. Please summarize the issues you discovered in the Health Risk Assessment Protocol**  
13 **and the Health Risk Assessment Report.**

14 **A.** I also reviewed the Health Risk Assessment Protocol (HRAP) and Health Risk Assessment  
15 Report (HRA) submitted to RIDEM. The HRAP is a document which describes the  
16 methodology which will be used to assess human health risk which is then documented in  
17 the HRA. Restated, the HRAP is the “roadmap” of the assessment, whereas the HRA is the  
18 actual “findings” of the assessment. These documents also had inconsistencies and  
19 omissions. These included:

- 20 • Assumptions related to ULSD fuel use and consumption that were undefined;
- 21 • Assumptions related to modeled pollutants that were not defined or referenced;
- 22 • Sensitive receptor information was missing from HRAP;
- 23 • Risk Exposure Scenario Assumptions were not defined or referenced.
- 24



1  
2 Although the HRA was not wholly revised, several tables, figures, and charts were updated  
3 and included as Attachment 2 to the Major Source Permit Application Addendum. A  
4 revised Figure 6 (Maximally Impacted Receptors) of the HRA included as part of  
5 Attachment 2 of the Major Source Permit Application Addendum indicates significantly  
6 fewer Maximally Impacted Receptors than were identified in original HRA Figure 6. The  
7 basis of this reduction or the methodology of the receptor analysis is not adequately  
8 explained.

9  
10 Additionally, the revised Table 1, Facility Potential Emission Summary, contains an  
11 additional column for Gas Turbines/HRSG/Duct Burner Steady State Operations,  
12 identified as "N.Gas w/DB". This may represent natural gas firing with duct burners.  
13 However, it is not clear if this scenario was not considered in previous permit application  
14 materials, or if this was the previous application assumption. Additional clarification is  
15 needed to distinguish this from other "N.Gas" column, to determine what scenario was  
16 previously considered in 2015 application, and what facility information triggered this  
17 additional column/information. These inconsistencies need to be explained and adequately  
18 addressed before a full review can be made. Otherwise, the HRA, even with amended  
19 tables/figures/charts, is incomplete.

## 20 21 EMISSION REDUCTION CREDITS

22 **Q. Will Burrillville benefit from the Emission Reduction Credits (ERC)?**

23 A. No. Within the Major Source Permit Application and in an Emission Offset

24 Correspondence dated August 28, 2015, both prepared by ESS, ESS has stated that there

1 may be some benefits in the generation of ERC. However, the proposed ERC which  
2 Invenergy hopes to obtain represents emissions originating in Saratoga Springs, NY, i.e., a  
3 pollution emitting facility is being shuttered in Saratoga Springs, NY and will cease  
4 emissions. . [It is worth noting that the noted 2015 ESS correspondence references  
5 “Sarasota Springs, NY”, as the source of the ERC, which we have assumed to be Saratoga  
6 Springs, NY.] The CREC located in Burrillville, RI will “obtain” these credits and be  
7 allowed to emit increased amounts of air pollutants in Rhode Island. Therefore, the ERC  
8 offer no benefit at all to the local air quality. In fact, they may represent a significant  
9 disadvantage to the air quality Rhode Island and Burrillville.

11 **CARBON DIOXIDE (CO2) SEQUESTRATION,**

12 **OXYGEN (O2) PRODUCTION, AND AIR EMISSIONS**

13 **Q. Art there air quality benefits to leaving the CREC site undeveloped?**

14 A. Absolutely. Forested land offers two major benefits to air quality. Woodlands sequester  
15 CO<sub>2</sub> and emit oxygen. CO<sub>2</sub>, which is a greenhouse gas, a major cause of global warming,  
16 and a criteria air pollutant, is absorbed by forested land at a rate of between one (1) and two  
17 (2) tons per acre per year, although estimates vary depending on tree species, tree density,  
18 soil type, etc. When forests are cleared, both of these benefits are greatly diminished.

20 **Q. What will be the disadvantages to air quality if the CREC facility is constructed and  
21 operated as proposed?**

22 A. It is my professional opinion that the CREC facility will cause an unacceptable harm to the  
23 environmental, particularly the purity of the air, the health of citizens, and the aesthetic value  
24 of the area. The proposed CREC facility will emit significant amounts of many air

1 pollutants, including: sulfur dioxide (SO<sub>2</sub>); particulate matter (PM), carbon monoxide (CO),  
2 carbon dioxide (CO<sub>2</sub>) ozone, oxides of nitrogen (NO<sub>x</sub>), volatile organic compounds (VOCs),  
3 known as criteria air pollutants, and many other pollutants. The facility will also emit various  
4 Hazardous Air Pollutants (HAPs). On the table attached here as *Exhibit EE1*, I have  
5 summarized the Potential to Emit (PTE) taken from the Major Source Permit Application  
6 Addendum prepared by ESS and dated September 15, 2016 and the health impacts of each  
7 anticipated pollutant. Per ESS's estimates of each pollutant's annual PTE, the facility would  
8 have the potential to emit the following amounts of criteria pollutants **annually** if  
9 constructed as proposed:

10 546,000 pounds of NO<sub>x</sub>;  
11 446,000 pounds of CO;  
12 156,000 pounds of VOCs;  
13 4.6 pounds of lead;  
14 310,000 pounds of PM;  
15 104,000 pounds of SO<sub>2</sub>; and,  
16 7,208,712,000 pounds of CO<sub>2</sub>.

17 Further, the facility would have the potential to emit the following amounts of HAPs  
18 **annually** if constructed as proposed:

19 1,700 pounds of hexane;  
20 1,370 pounds of formaldehyde;  
21 755 pounds of toluene;  
22 370 pounds of xylenes;  
23 232 pounds of acetaldehyde;  
24 185 pounds of ethylbenzene;

1 167 pounds of propylene oxide;  
2 90.8 pounds of benzene;  
3 37 pounds of acrolein;  
4 18.1 pounds of naphthalene;  
5 15 pounds of nickel;  
6 13.3 pounds of chromium;  
7 6.73 pounds of 1,3-Butadiene;  
8 5.8 pounds of cadmium;  
9 2.75 pounds of manganese;  
10 1.8 pounds of arsenic;  
11 1.4 pounds of mercury;  
12 0.88 pounds of beryllium;  
13 0.8 pounds of selenium;  
14 0.4 pounds of chromium; and,  
15 0.4 pounds of cobalt.

16 Please refer to the attached *Exhibit EE1*, which details the potential amount of each criteria  
17 air pollutant and HAP which may be emitted by the proposed facility. *Exhibit EE1* further  
18 details the possible acute and chronic health effects of each pollutant.

19  
20 Each of the emitted pollutants listed above have both acute and chronic negative health  
21 effects on animals and humans, although the effects are varied in nature and degree. Of the  
22 identified HAPs, 1,3- butadiene, arsenic, benzene, and chromium are known human  
23 carcinogens, while acetaldehyde, beryllium, cadmium, formaldehyde, naphthalene, some  
24 nickel forms, and propylene oxide are probable or possible carcinogens. NO<sub>x</sub>, VOCs, lead,

1 PM, SO<sub>2</sub>, 1,3-butadiene, acetaldehyde, acrolein, benzene, beryllium, cadmium, chromium,  
2 cobalt, ethylbenzene, formaldehyde, nickel, propylene oxide, selenium, toluene, and xylenes  
3 are documented to have adverse effects on the respiratory system, particularly harming those  
4 with immune deficiencies or sensitive populations like the elderly or children. These  
5 respiratory-affecting compounds would cause or exacerbate breathing problems like asthma,  
6 Chronic Obstructive Pulmonary Disease (COPD), decreased lung function, and irritation to  
7 airways. PM, NO<sub>x</sub>, and SO<sub>2</sub> contribute to acid rain, which can irreversibly harm sensitive  
8 ecosystems and waterbodies. Other pollutants can damage natural ecosystems, adversely  
9 affect cropland, and contribute to nutrient pollution in wetlands and waterbodies.

10  
11 If the facility were not constructed as proposed, these potential adverse health and  
12 environmental effects on the citizens for Rhode Island and Burrillville would be eliminated.

13  
14 **Q. You stated you reviewed the Revised Water Supply Plan prepared by ESS and dated**  
15 **January 11, 2017. Does this plan have negative air quality implications?**

16 A. Yes. The proposed method for supplying the facility with process water includes trucking  
17 the water from the Town of Johnson, Rhode Island as its primary supply, and trucking from  
18 other alternate water suppliers as a contingency plan. Based on information in the revised  
19 Water Supply Plan, the facility will require more than 720,000 gallons of process water per  
20 day when firing one gas turbine using ULSD, although the report states that typical water  
21 usage would be between 15,000 and 19,000 gallons per day. The movement on/off site of  
22 water trailers, demineralization trailers, the offsite disposal of generated wastewater, and the  
23 delivery of fuel oil, ammonia, and other supplies and material also adds truck traffic to the  
24 project area. After an oil fired event, the traffic analysis predicts that approximately 22 trucks

1 per day will access the site. When considering typical daily trucking requirements for process  
2 water and waste transport, in addition to the significant increase in water needs and truck  
3 traffic when oil firing occurs, the continuous truck traffic results in an increase in air  
4 emissions from trucks to the project area and beyond. Diesel emissions have been  
5 demonstrated to contribute to adverse local health and environmental impacts.

6  
7 **Q. Mr. Michael Feinblatt of ESS contends that the construction of the CREC facility**  
8 **will ultimately lead to decreases in air emissions. Do you agree?**

9 A. No. Air emissions in Rhode Island and Burrillville will greatly increase. For example, over  
10 7.2 billion pounds of carbon dioxide could be emitted from the CREC facility annually, an  
11 increase of 30% over existing Rhode Island carbon dioxide emissions. This will in my  
12 opinion make it virtually impossible for Rhode Island to comply with the Resilient Rhode  
13 Island Act and the Paris Agreement on global warming. As for whether regional air  
14 emissions would be reduced, there is simply not enough information to make such a claim.  
15 There are no guarantees, agreements, or binding contracts which would require older, less  
16 efficient generating facilities elsewhere in New England to be shut down when and if CREC  
17 is constructed.

18  
19 **EFFICIENCY OF SELECTED GE TURBINES**

20 **Q. It was noted within Mr. Mark Wiitanen's June 30, 2017 Pre-Filed Direct Testimony**  
21 **that the recently selected turbine equipment is the "most efficient" equipment**  
22 **available on the market today. Can you speak to this assertion?**

23 A. No. No information related to the efficiency of the selected General Electric (GE) model  
24 7HA.02 combustion turbine, as it relates to other commercially available turbines, has been

1 provided to me for review. Mr. Wiitanen also asserts that this will be the “most efficient  
2 power plant in New England”. I cannot affirm this statement either, because no  
3 substantiating information or references have been provided to me.  
4

## 5 CONCLUSION

6 **Q. What does all this mean?**

7 A. The modeling completed by Invenenergy and submitted to RIDEM claims to demonstrate that  
8 the maximum predicted impacts for the proposed facility will not cause or contribute to air  
9 pollution in violation of the National Ambient Air Quality Standards (NAAQS) for any of  
10 the scenarios considered. The NAAQS are standards established by the US Environmental  
11 Protection Agency under authority of the Clean Air Act that apply to outdoor air throughout  
12 the country. Primary standards are designed to protect human health, including sensitive  
13 populations such as children, the elderly, and individuals suffering from respiratory diseases.  
14 Secondary standards are designed to protect public welfare from any known or anticipated  
15 adverse effects of a pollutant. However, this conclusion is based on the unproven  
16 assumption that the neighboring Algonquin Compressor Station will make significant  
17 changes to its operations. Therefore, the Air Dispersion Modeling Report and Multisource  
18 Modeling Report cannot be accepted. Actually, I cannot confirm whether the proposed  
19 facility will cause or contribute to air pollution in violation of the NAAQS. That said, the  
20 facility will contribute to a significant increase in the levels of criteria pollutants surrounding  
21 the site. Emitted pollutants include sulfur dioxide (SO<sub>2</sub>), particulate matter (PM), carbon  
22 monoxide (CO), carbon dioxide (CO<sub>2</sub>) ozone, oxides of nitrogen (NO<sub>x</sub>), volatile organic  
23 compounds (VOCs), and various Hazardous Air Pollutants (HAPs). Each predicted air  
24 pollutant represents adverse impacts to human health and/or the environment. For

1 Burrillville residents, these emissions represent a significant increase in air pollution, since  
2 facility is a new source, not a replacement or modified facility. The CREC facility represents,  
3 in my professional opinion, a potential significant risk to the environment and the health of  
4 citizens of Burrillville and Rhode Island.

5  
6 **RECOMENDATION**

7 **Q. Are you recommending against the siting of the CREC within the Town of**  
8 **Burrillville?**

9 A. Yes. I am firmly of the opinion that the disadvantages of the proposed Invenergy facility  
10 greatly outweigh any potential benefits.

11  
12 **Q. Are the opinions you have expressed in your testimony based upon your education,**  
13 **training, experience and the materials you have reviewed to prepare for this**  
14 **testimony, and are those opinions all based upon a reasonable degree of certainty or**  
15 **probability in your fields of expertise?**

16 A. Yes.

17  
18 **Q. Does this conclude your testimony?**

19 A. Yes.



Clear River Energy Center  
 Summary of Potential Air Emissions and Effects  
 Prepared for Town of Burrillville, RI

Pollutant	Potential to Emit (lb/year)	Potential to Emit (TPY)	Information on Pollutant
<p><b>Criteria Air Pollutants</b> : EPA has established national ambient air quality standards (NAAQS) for six of the most common air pollutants—carbon monoxide, lead, ground-level ozone, particulate matter, nitrogen dioxide, and sulfur dioxide—known as “criteria” air pollutants (or simply “criteria pollutants”). The presence of these pollutants in ambient air is generally due to numerous diverse and widespread sources of emissions. The primary NAAQS are set to protect public health. EPA also sets secondary NAAQS to protect public welfare from adverse effects of criteria pollutants, including protection against visibility impairment, or damage to animals, crops, vegetation, or buildings. As required by the Clean Air Act, EPA periodically conducts comprehensive reviews of the scientific literature on health and welfare effects associated with exposure to the criteria air pollutants. The resulting assessments serve as the basis for making regulatory decisions about whether to retain or revise the NAAQS that specify the allowable concentrations of each of these pollutants in the ambient air.</p>			
Nitrogen Oxides (NOx)	546,000	273	<p>Precursor to ozone (project site is designated as moderate non-attainment area for ozone per the National Ambient Air Quality Standards, NAAQS). See effects of increased ozone levels below associated with VOCs.</p> <p>Health Effects: Breathing air with a high concentration of NO2 can irritate airways in the human respiratory system. Such exposures over short periods can aggravate respiratory diseases, particularly asthma, leading to respiratory symptoms (such as coughing, wheezing or difficulty breathing), hospital admissions and visits to emergency rooms. Longer exposures to elevated concentrations of NO2 may contribute to the development of asthma and potentially increase susceptibility to respiratory infections. People with asthma, as well as children and the elderly are generally at greater risk for the health effects of NO2. NO2 along with other NOx reacts with other chemicals in the air to form both particulate matter and ozone. Both of these are also harmful when inhaled due to effects on the respiratory system.</p> <p>Environmental Effects: NO2 and other NOx interact with water, oxygen and other chemicals in the atmosphere to form acid rain. Acid rain harms sensitive ecosystems such as lakes and forests. The nitrate particles that result from NOx make the air hazy and difficult to see through. This affects the many national parks that we visit for the view. NOx in the atmosphere contributes to nutrient pollution in coastal waters.</p>
Carbon Monoxide (CO)	446,000	223	<p>Health Effects: Breathing air with a high concentration of CO reduces the amount of oxygen that can be transported in the blood stream to critical organs like the heart and brain. When CO levels are elevated outdoors, they can be of particular concern for people with some types of heart disease. These people already have a reduced ability for getting oxygenated blood to their hearts in situations where the heart needs more oxygen than usual. They are especially vulnerable to the effects of CO when exercising or under increased stress. In these situations, short-term exposure to elevated CO may result in reduced oxygen to the heart accompanied by chest pain also known as angina.</p>
Volatile Organic Compounds (VOC)	156,000	78	<p>Precursor to ozone (project site is designated as moderate non-attainment area for ozone per the NAAQS). Ground level or “bad” ozone is not emitted directly into the air, but is created by chemical reactions between oxides of nitrogen (NOx) and volatile organic compounds (VOC) in the presence of sunlight.</p> <p>Health Effects of Ozone: People most at risk from breathing air containing ozone include people with asthma, children, older adults, and people who are active outdoors, especially outdoor workers. In addition, people with certain genetic characteristics, and people with reduced intake of certain nutrients, such as vitamins C and E, are at greater risk from ozone exposure. Breathing ozone can trigger a variety of health problems including chest pain, coughing, throat irritation, and airway inflammation. It also can reduce lung function and harm lung tissue. Ozone can worsen bronchitis, emphysema, and asthma, leading to increased medical care.</p> <p>Environmental Effects of Ozone: Ozone affects sensitive vegetation and ecosystems, including forests, parks, wildlife refuges and wilderness areas. In particular, ozone harms sensitive vegetation during the growing season.</p>
Lead	4.6	2.3E-03	<p>Health Effects: Once taken into the body, lead distributes throughout the body in the blood and is accumulated in the bones. Depending on the level of exposure, lead can adversely affect the nervous system, kidney function, immune system, reproductive and developmental systems and the cardiovascular system. Lead exposure also affects the oxygen carrying capacity of the blood. The lead effects most commonly encountered in current populations are neurological effects in children and cardiovascular effects (e.g., high blood pressure and heart disease) in adults. Infants and young children are especially sensitive to even low levels of lead, which may contribute to behavioral problems, learning deficits and lowered IQ.</p> <p>Environmental Effects: Lead is persistent in the environment and can be added to soils and sediments through deposition from sources of lead air pollution. Other sources of lead to ecosystems include direct discharge of waste streams to water bodies and mining. Elevated lead in the environment can result in decreased growth and reproductive rates in plants and animals, and neurological effects in vertebrates.</p>

Clear River Energy Center  
 Summary of Potential Air Emissions and Effects  
 Prepared for Town of Burrillville, RI

Pollutant	Potential to Emit (lb/year) <sup>1</sup>	Potential to Emit (TPY) <sup>1</sup>	Information on Pollutant
Particulate Matter (PM/PM10/PM2.5)	310,000	155	<p><b>Health Effects:</b> The size of particles is directly linked to their potential for causing health problems. Small particles less than 10 micrometers in diameter pose the greatest problems, because they can get deep into your lungs, and some may even get into your bloodstream. Exposure to such particles can affect both the lungs and the heart. Numerous scientific studies have linked particle pollution exposure to a variety of problems, including: premature death in people with heart or lung disease, nonfatal heart attacks, irregular heartbeat, aggravated asthma, decreased lung function, increased respiratory symptoms, such as irritation of the airways, coughing or difficulty breathing. People with heart or lung diseases, children, and older adults are the most likely to be affected by particle pollution exposure</p> <p><b>Environmental Effects:</b> Fine particles (PM2.5) are the main cause of reduced visibility (haze) in parts of the United States. Particles can be carried over long distances by wind and then settle on ground or water. Depending on their chemical composition, the effects of this settling may include making lakes and streams acidic, changing the nutrient balance in coastal waters and large river basins, depleting the nutrients in soil, damaging sensitive forests and farm crops, affecting the diversity of ecosystems, and contributing to acid rain effects.</p>
Sulfur Dioxide (SO2)	104,000	52	<p><b>Health Effects:</b> Short-term exposures to SO2 can harm the human respiratory system and make breathing difficult. Children, the elderly, and those who suffer from asthma are particularly sensitive to effects of SO2. SO2 emissions that lead to high concentrations of SO2 in the air generally also lead to the formation of other sulfur oxides (SOx). SOx can react with other compounds in the atmosphere to form small particles. These particles contribute to particulate matter (PM) pollution: particles may penetrate deeply into sensitive parts of the lungs and cause additional health problems.</p> <p><b>Environmental Effects:</b> At high concentrations, gaseous SOx can harm trees and plants by damaging foliage and decreasing growth. SO2 and other sulfur oxides can contribute to acid rain which can harm sensitive ecosystems.</p>
Carbon Dioxide (CO2)	7,208,712,000	3,604,356	<p>Although not a true criteria air pollutant, CO2 is closely monitored as it is the leading greenhouse gas. Gases that trap heat in the atmosphere are called greenhouse gases. Carbon dioxide (CO2) is the primary greenhouse gas emitted through human activities. In 2014, CO2 accounted for about 80.9% of all U.S. greenhouse gas emissions from human activities. Carbon dioxide is naturally present in the atmosphere as part of the Earth's carbon cycle. Human activities are altering the carbon cycle—both by adding more CO2 to the atmosphere and by influencing the ability of natural sinks, like forests, to remove CO2 from the atmosphere. While CO2 emissions come from a variety of natural sources, human-related emissions are responsible for the increase that has occurred in the atmosphere since the industrial revolution. The main human activity that emits CO2 is the combustion of fossil fuels (coal, natural gas, and oil) for energy and transportation, although certain industrial processes and land-use changes also emit CO2.<sup>5</sup></p>
<p><b>Hazardous Air Pollutants (HAP):</b> Hazardous air pollutants are those known to cause cancer and other serious health impacts. The Clean Air Act requires the EPA to regulate toxic air pollutants, also known as air toxics.<sup>4</sup></p>			
1,3-Butadiene	6.73	3.4E-03	<p>Acute (short-term) exposure to 1,3-butadiene by inhalation in humans results in irritation of the eyes, nasal passages, throat, and lungs. Epidemiological studies have reported a possible association between 1,3-butadiene exposure and cardiovascular diseases. Epidemiological studies have shown an association between 1,3-butadiene exposure and increased incidence of leukemia. Animal studies have reported tumors at various sites from 1,3-butadiene exposure. EPA has classified 1,3-butadiene as carcinogenic to humans by inhalation.</p>
Acetaldehyde	232	1.2E-01	<p>Acute (short-term) exposure to acetaldehyde results in effects including irritation of the eyes, skin, and respiratory tract. Symptoms of chronic (long-term) intoxication of acetaldehyde resemble those of alcoholism. Acetaldehyde is considered a probable human carcinogen (Group B2) based on inadequate human cancer studies and animal studies that have shown nasal tumors in rats and laryngeal tumors in hamsters.</p>
Acrolein	37	1.9E-02	<p>Acrolein may be formed from the breakdown of certain pollutants in outdoor air or from the burning of organic matter including tobacco, or fuels such as gasoline or oil. It is toxic to humans following inhalation, oral or dermal exposures. Acute (short-term) inhalation exposure may result in upper respiratory tract irritation and congestion. No information is available on its reproductive, developmental, or carcinogenic effects in humans, and the existing animal cancer data are considered inadequate to make a determination that acrolein is carcinogenic to humans.</p>

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Arsenic	1.18	5.9E-04	For most people, food is the major source of arsenic exposure. Acute (short-term) high-level inhalation exposure to arsenic dust or fumes has resulted in gastrointestinal effects (nausea, diarrhea, abdominal pain); central and peripheral nervous system disorders have occurred in workers acutely exposed to inorganic arsenic. Chronic (long-term) inhalation exposure to inorganic arsenic is associated with irritation of the skin and mucous membranes and effects in the brain and nervous system. Chronic oral exposure to elevated levels of inorganic arsenic has resulted in gastrointestinal effects, anemia, peripheral neuropathy, skin lesions, hyperpigmentation, and liver or kidney damage in humans. Inorganic arsenic exposure of humans, by the inhalation route, has been shown to be strongly associated with lung cancer, while ingestion of inorganic arsenic by humans has been linked to a form of skin cancer and also to bladder, liver, and lung cancer. EPA has classified inorganic arsenic as a human carcinogen.
Benzene	90.8	4.5E-02	Benzene is found in the air from emissions from burning coal and oil, gasoline service stations, and motor vehicle exhaust. Acute (short-term) inhalation exposure of humans to benzene may cause drowsiness, dizziness, headaches, as well as eye, skin, and respiratory tract irritation, and, at high levels, unconsciousness. Chronic (long-term) inhalation exposure has caused various disorders in the blood, including reduced numbers of red blood cells and aplastic anemia, in occupational settings. Reproductive effects have been reported for women exposed by inhalation to high levels, and adverse effects on the developing fetus have been observed in animal tests. Increased incidence of leukemia (cancer of the tissues that form white blood cells) have been observed in humans occupationally exposed to benzene. EPA has classified benzene as known human carcinogen for all routes of exposure.
Beryllium	0.88	4.4E-04	Inhalation exposure to beryllium primarily occurs from the burning of coal or fuel oil and in tobacco smoke. Acute (short-term) inhalation exposure to high levels of beryllium has been observed to cause inflammation of the lungs or acute pneumonitis (reddening and swelling of the lungs) in humans; after exposure ends, these symptoms may be reversible. Chronic (long-term) inhalation exposure of humans to beryllium has been reported to cause chronic beryllium disease (berylliosis), in which granulomatous lesions (noncancerous) develop in the lung. Human epidemiology studies are limited, but suggest a causal relationship between beryllium exposure and an increased risk of lung cancer. Inhalation exposure to beryllium has been demonstrated to cause lung cancer in rats and monkeys. EPA has classified beryllium as a Group B1, probable human carcinogen.
Cadmium	5.8	2.9E-03	The main sources of cadmium in the air are the burning of fossil fuels such as coal or oil and the incineration of municipal waste. The acute (short-term) effects of cadmium in humans through inhalation exposure consist mainly of effects on the lung, such as pulmonary irritation. Chronic (long-term) inhalation or oral exposure to cadmium leads to a build-up of cadmium in the kidneys that can cause kidney disease. Cadmium has been shown to be a developmental toxicant in animals, resulting in fetal malformations and other effects, but no conclusive evidence exists in humans. An association between cadmium exposure and an increased risk of lung cancer has been reported from human studies, but these studies are inconclusive due to confounding factors. Animal studies have demonstrated an increase in lung cancer from long-term inhalation exposure to cadmium. EPA has classified cadmium as a Group B1, probable human carcinogen.
Chromium	13.3	6.7E-03	Chromium occurs in the environment primarily in two valence states, trivalent chromium (Cr III) and hexavalent chromium (Cr VI). Exposure may occur from natural or industrial sources of chromium. Chromium III is much less toxic than chromium (VI). The respiratory tract is also the major target organ for chromium (III) toxicity, similar to chromium (VI). Chromium (III) is an essential element in humans. The body can detoxify some amount of chromium (VI) to chromium (III). The respiratory tract is the major target organ for chromium (VI) toxicity, for acute (short-term) and chronic (long-term) inhalation exposures. Shortness of breath, coughing, and wheezing were reported from a case of acute exposure to chromium (VI), while perforations and ulcerations of the septum, bronchitis, decreased pulmonary function, pneumonia, and other respiratory effects have been noted from chronic exposure. Human studies have clearly established that inhaled chromium (VI) is a human carcinogen, resulting in an increased risk of lung cancer. Animal studies have shown chromium (VI) to cause lung tumors via inhalation exposure.
Cobalt	0.44	2.2E-04	Cobalt is a natural element found throughout the environment. Acute (short-term) exposure to high levels of cobalt by inhalation in humans and animals results in respiratory effects, such as a significant decrease in ventilatory function, congestion, edema, and hemorrhage of the lung. Respiratory effects are also the major effects noted from chronic (long-term) exposure to cobalt by inhalation, with respiratory irritation, wheezing, asthma, pneumonia, and fibrosis noted. Cardiac effects, congestion of the liver, kidneys, and conjunctiva, and immunological effects have also been noted in chronically-exposed humans. Cobalt is an essential element in humans, as a constituent of vitamin B12. Human studies are inconclusive regarding inhalation exposure to cobalt and cancer, and the one available oral study did not report a correlation between cobalt in the drinking water and cancer deaths. EPA has not classified cobalt for carcinogenicity.

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Ethylbenzene	185	9.3E-02	Acute (short-term) exposure to ethylbenzene in humans results in respiratory effects, such as throat irritation and chest constriction, irritation of the eyes, and neurological effects such as dizziness. Chronic (long-term) exposure to ethylbenzene by inhalation in humans has shown conflicting results regarding its effects on the blood, liver, and kidneys from chronic inhalation exposure to ethylbenzene. Limited information is available on the carcinogenic effects of ethylbenzene in humans. In a study by the National Toxicology Program (NTP), exposure to ethylbenzene by inhalation resulted in an increased incidence of kidney and testicular tumors in rats, and lung and liver tumors in mice. EPA has classified ethylbenzene as a Group D, not classifiable as to human carcinogenicity.
Formaldehyde	1,370	6.9E-01	Exposure to formaldehyde may occur by breathing contaminated indoor air, tobacco smoke, or ambient urban air. Acute (short-term) and chronic (long-term) inhalation exposure to formaldehyde in humans can result in respiratory symptoms, and eye, nose, and throat irritation. Limited human studies have reported an association between formaldehyde exposure and lung and nasopharyngeal cancer. Animal inhalation studies have reported an increased incidence of nasal squamous cell cancer. EPA considers formaldehyde a probable human carcinogen (Group B1).
Hexane	1,700	8.5E-01	Acute (short-term) inhalation exposure of humans to high levels of hexane causes mild central nervous system (CNS) effects, including dizziness, giddiness, slight nausea, and headache. Chronic (long-term) exposure to hexane in air is associated with polyneuropathy in humans, with numbness in the extremities, muscular weakness, blurred vision, headache, and fatigue observed. Neurotoxic effects have also been exhibited in rats. No information is available on the carcinogenic effects of hexane in humans or animals. EPA has classified hexane as a Group D, not classifiable as to human carcinogenicity.
Manganese	2.75	1.4E-03	Manganese is naturally occurring in the environment. Manganese is essential for normal physiologic functioning in humans and animals, and exposure to low levels of manganese in the diet is considered to be nutritionally essential in humans. Chronic (long-term) exposure to high levels of manganese by inhalation in humans may result in central nervous system (CNS) effects. Visual reaction time, hand steadiness, and eye-hand coordination were affected in chronically-exposed workers. A syndrome named manganism may result from chronic exposure to higher levels; manganism is characterized by feelings of weakness and lethargy, tremors, a mask-like face, and psychological disturbances. Respiratory effects have also been noted in workers chronically exposed to manganese bearing particles by inhalation.
Mercury	1.4	7.0E-04	Mercury exists in three forms: elemental mercury, inorganic mercury compounds (primarily mercuric chloride), and organic mercury compounds (primarily methyl mercury). All forms of mercury are quite toxic, and each form exhibits different health effects.  Acute (short-term) exposure to high levels of elemental mercury in humans results in central nervous system (CNS) effects such as tremors, mood changes, and slowed sensory and motor nerve function. Chronic (long-term) exposure to elemental mercury in humans also affects the CNS, with effects such as erethism (increased excitability), irritability, excessive shyness, and tremors. Human studies are inconclusive regarding elemental mercury and cancer. Acute exposure to inorganic mercury by the oral route may result in effects such as nausea, vomiting, and severe abdominal pain. The major effect from chronic exposure to inorganic mercury is kidney damage. Animal studies have reported effects such as alterations in testicular tissue, increased resorption rates, and abnormalities of development. Mercuric chloride (an inorganic mercury compound) exposure has been shown to result in forestomach, thyroid, and renal tumors in experimental animals.  Acute exposure of humans to very high levels of methyl mercury results in CNS effects such as blindness, deafness, and impaired level of consciousness. Chronic exposure to methyl mercury in humans also affects the CNS with symptoms such as paresthesia (a sensation of pricking on the skin), blurred vision, malaise, speech difficulties, and constriction of the visual field. Methyl mercury exposure, via the oral route, has led to significant developmental effects. Infants born to women who ingested high levels of methyl mercury exhibited mental retardation, ataxia, constriction of the visual field, blindness, and cerebral palsy.
Naphthalene	18.1	9.1E-03	Acute (short-term) exposure of humans to naphthalene by inhalation, ingestion, and dermal contact is associated with hemolytic anemia, damage to the liver, and neurological damage. Cataracts have also been reported in workers acutely exposed to naphthalene by inhalation and ingestion. Chronic (long-term) exposure of workers and rodents to naphthalene has been reported to cause cataracts and damage to the retina. Available data are inadequate to establish a causal relationship between exposure to naphthalene and cancer in humans. EPA has classified naphthalene as a Group C, possible human carcinogen.

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Nickel	15	7.5E-03	Nickel occurs naturally in the environment at low levels. Nickel is an essential element in some animal species, and it has been suggested it may be essential for human nutrition. Nickel dermatitis, consisting of itching of the fingers, hands, and forearms, is the most common effect in humans from chronic (long-term) skin contact with nickel. Respiratory effects have also been reported in humans from inhalation exposure to nickel. Human and animal studies have reported an increased risk of lung and nasal cancers from exposure to nickel refinery dusts and nickel subsulfide. Animal studies of soluble nickel compounds (i.e., nickel carbonyl) have reported lung tumors. EPA has classified nickel refinery dust and nickel subsulfide as Group A, human carcinogens, and nickel carbonyl as a Group B2, probable human carcinogen.
Propylene Oxide	167	8.4E-02	Acute (short-term) exposure of humans and animals to propylene oxide has caused eye and respiratory tract irritation. Dermal contact, even with dilute solutions, has caused skin irritation and necrosis in humans. Propylene oxide is also a mild central nervous system (CNS) depressant in humans. Inflammatory lesions of the nasal cavity, trachea, and lungs and neurological effects have been observed in animals chronically (long-term) exposed to propylene oxide by inhalation. Propylene oxide has been observed to cause tumors at or near the site of administration in rodents, causing forestomach tumors following ingestion via gavage (experimentally placing the chemical in the stomach) and nasal tumors after inhalation exposure. EPA has classified propylene oxide as a Group B2, probable human carcinogen.
Selenium	0.8	4.0E-04	Selenium is a naturally occurring substance that is toxic at high concentrations but is also a nutritionally essential element. Hydrogen selenide is the most acutely toxic selenium compound. Acute (short-term) exposure to elemental selenium, hydrogen selenide, and selenium dioxide by inhalation results primarily in respiratory effects, such as irritation of the mucous membranes, pulmonary edema, severe bronchitis, and bronchial pneumonia. Epidemiological studies of humans chronically (long-term) exposed to high levels of selenium in food and water have reported discoloration of the skin, pathological deformation and loss of nails, loss of hair, excessive tooth decay and discoloration, lack of mental alertness, and listlessness. The only selenium compound that has been shown to be carcinogenic in animals is selenium sulfide, which resulted in an increase in liver tumors from oral exposure. EPA has classified elemental selenium as a Group D, not classifiable as to human carcinogenicity, and selenium sulfide as a Group B2, probable human carcinogen.
Toluene	755	3.8E-01	The central nervous system (CNS) is the primary target organ for toluene toxicity in both humans and animals for acute (short-term) and chronic (long-term) exposures. CNS dysfunction and narcosis have been frequently observed in humans acutely exposed to elevated airborne levels of toluene; symptoms include fatigue, sleepiness, headaches, and nausea. CNS depression has been reported to occur in chronic abusers exposed to high levels of toluene. Chronic inhalation exposure of humans to toluene also causes irritation of the upper respiratory tract and eyes, sore throat, dizziness, and headache. Human studies have reported developmental effects, such as CNS dysfunction, attention deficits, and minor craniofacial and limb anomalies, in the children of pregnant women exposed to high levels of toluene or mixed solvents by inhalation. EPA has concluded that there is inadequate information to assess the carcinogenic potential of toluene.
Xylenes	370	1.9E-01	Xylenes are released into the atmosphere as fugitive emissions from industrial sources, from auto exhaust, and through volatilization from their use as solvents. Acute (short-term) inhalation exposure to mixed xylenes in humans results in irritation of the eyes, nose, and throat, gastrointestinal effects, eye irritation, and neurological effects. Chronic (long-term) inhalation exposure of humans to mixed xylenes results primarily in central nervous system (CNS) effects, such as headache, dizziness, fatigue, tremors, and incoordination; respiratory, cardiovascular, and kidney effects have also been reported. EPA has classified mixed xylenes as a Group D, not classifiable as to human carcinogenicity.

Notes:

- 1: Potential Emission estimates taken from the Major Source Permit Application Addendum , Combined-Cycle Electric Generating Facility, prepared by ESS Group, dated September 15, 2016.
- 2: Criteria air pollutant designated per the National Ambient Air Quality Standards (NAAQS) of the Clean Air Act (CAA).
- 3: Information taken from EPA Criteria Pollutant Website (<https://www.epa.gov/criteria-air-pollutants>).
- 4: Information taken from EPA HAP Website and factsheets (<https://www.epa.gov/haps/health-effects-notebook-hazardous-air-pollutants>).
- 5: Information taken from EPA's Overview of Greenhouse Gases Webpage, Carbon Dioxide (<https://www.epa.gov/ghgemissions/overview-greenhouse-gases/>)  
 Other air constituents will also be emitted by the proposed facility, however, air constituents that are not listed as a criteria air pollutant or a HAP were omitted from this table (i.e., this is not an exclusive list of emitted compounds).