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BEFORE THE STATE OF WASHINGTON  
ENERGY FACILITY SITE EVALUATION COUNCIL

In the Matter of Application No. 99-1:

SUMAS ENERGY 2 GENERATION  
FACILITY

**EXHIBIT \_\_\_\_ (SP-RT)**

**APPLICANT'S PRE-FILED REBUTTAL TESTIMONY**

**WITNESS: SANYA PETROVIC**

**Q. Please re-introduce yourself to the Council.**

A. My name is Sanya Petrovic. I am Group Manager for Risk Assessment at the consulting firm Jacques Whitford Environment Limited located in Burnaby, British Columbia.

1 **Q. What issues will you address in this rebuttal testimony?**

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3 A. Like my pre-filed direct testimony, my rebuttal testimony will address issues related  
4 to the potential health impacts of air emissions from the modified SE2 proposal.  
5 Specifically, I have reviewed the testimony of Robert Caton, David Bates and  
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7 Michael Lepage submitted by the Province of British Columbia, as well as the  
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9 testimony of Jane Koenig submitted by Whatcom County, and my rebuttal testimony  
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11 responds to portions of the testimony provided by those witnesses.  
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17 **Q. You said you read Robert Caton’s testimony. What was your general reaction to**  
18 **his testimony?**

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20 A. It read more like a lawyer’s legal brief rather than a scientist’s testimony. It seemed to  
21  
22 be more focused on Dr. Caton’s understanding of the rules related to this process than  
23  
24 on the science at issue. He quoted the Council’s previous decision repeatedly and  
25  
26 seemed almost to be trying to predict whether information in the Revised Application  
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28 would change the Council’s opinion. Significantly, Dr. Caton agrees with me that  
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30 “[t]he real issue is one of acceptable risk” (page 12, line 6). At the same time,  
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32 however, he never provides his professional opinion regarding what is considered to  
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34 be an acceptable level of risk.  
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39 **Q. What was your reaction to David Bates’ testimony?**

40 A. He frequently talked about whether the revised application provides a basis for the  
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42 Council to modify its earlier conclusions, and much of his testimony merely repeats  
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44 and paraphrases the testimony of other witnesses. The real substance of his testimony  
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1 is found in his discussion of "new" studies about health impacts, and I'd like to  
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3 address those specifically later.  
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7 **Q. What was your reaction to Michael Lepage's testimony?**

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9 A. Most of Mr. Lepage's testimony does not concern health effects per se. Rather, it  
10 addresses the calculation of the project's emissions and the modeling of ambient air  
11 concentrations. I have relied upon the data from Eric Hansen and MFG in this regard,  
12 and, for the most part, I will rely upon Mr. Hansen to respond to Mr. Lepage's  
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testimony.

**Q. What was your reaction to Jane Koenig's testimony?**

A. Dr. Koenig's testimony is very brief, referring back to her earlier testimony. She has  
made one particular point about particulate matter emissions, which I can address  
later.

#### Annual Average Ambient Air Quality

**Q. In their prefiled testimony, both Robert Caton and David Bates emphasize that the analysis of health effects should consider potential impacts from both long-term (annual) exposures and short-term (peak) exposures. Do you agree?**

A. Yes. The data in the Second Revised Application addresses the effects of the project's emissions on both annual average ambient concentrations and 24-hour maximum concentrations. My pre-filed direct testimony addressed the health implications of both annual average and 24-hour maximum incremental increases.

1 **Q. Let's talk about "long-term" or average annual concentrations first. Robert**  
2 **Caton states in his pre-filed testimony that "[b]ecause there is little change in the**  
3 **annual air pollution emissions from the facility, there is little change in the**  
4 **health impacts associated with those emissions." How do you respond to that**  
5 **statement?**  
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11 A. In several ways: First, this statement, like many others found in Dr. Caton's pre-filed  
12 testimony, is oddly phrased. He essentially says that IF you thought the project's  
13 annual emissions produced unacceptable health risks before, then you'll probably still  
14 think so, because the annual emissions haven't changed much.  
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21 Second, there is no reason to believe that there will be measurable adverse health  
22 effects attributable to the annual emissions from the revised SE2 project. In these  
23 proceedings, particulate matter and ozone are the ambient air quality issues that have  
24 been identified as concerns by some parties. Regarding particulate matter,  
25 conservative modeling of the annual SE2 emissions shows an annual average effect  
26 on air quality of only 0.38 ug/m<sup>3</sup> at the maximum point of impingement in Canada  
27 compared to background annual levels of around 15 ug/m<sup>3</sup> PM<sub>10</sub> and around 7 ug/m<sup>3</sup>  
28 of PM<sub>2.5</sub>. To put these numbers into perspective, the annual US NAAQS for PM<sub>10</sub> is  
29 50 ug/m<sup>3</sup>, the 24-hour British Columbia objective for PM<sub>10</sub> is 50 ug/m<sup>3</sup>, the annual  
30 GVRD objective for PM<sub>10</sub> is 30 ug/m<sup>3</sup> and the Canada-wide Standard for PM<sub>2.5</sub> is 30  
31 ug/m<sup>3</sup> (24-hour average, 98<sup>th</sup> percentile, averaged over 3 years). Based on available  
32 scientific literature, there is no evidence that a 0.38 ug/m<sup>3</sup> change in average annual  
33 concentrations would result in a measurable impact on public health. Regarding  
34 ozone, the Environment Canada ozone modeling (based on the higher emissions from  
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1 the original project) indicated that the SE2 emissions would rarely result in more than  
2 a 0.2 ppb increase in ozone concentrations in Abbotsford. No study has ever  
3 demonstrated a measurable impact to public health attributable to this small of a  
4 change in ozone levels.  
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10 Third, the modeling that Eric Hansen and MFG have performed demonstrates that the  
11 changes SE2 has made in the project – reducing emissions and raising the exhaust  
12 stack – have further reduced the project's effect on ambient air quality. This includes  
13 reductions in annual average as well as on short-term maximum concentrations of  
14 regulated pollutants attributable to SE2 emissions. Dr. Caton and Dr. Bates repeatedly  
15 emphasize that there are theoretical risks associated with any increases in air  
16 pollution. If the risks are linear, then the revised project's reduced impact on ambient  
17 air quality will result in a corresponding reduction in theoretical risk. I note,  
18 furthermore, that the revised Application and Eric Hansen's testimony indicate that  
19 the project changes result in a 40% reduction in NO<sub>2</sub> concentrations (NO<sub>x</sub> is a  
20 precursor to ozone) and a 10% reduction in particulate concentrations compared to the  
21 original application. It is my contention that the slight incremental increases in  
22 annual average ambient pollutant concentrations attributable to the revised SE2  
23 project will not result in measurable impacts to public health.  
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41 Finally, I must point out the apparent inconsistency in the testimony of Dr. Bates and  
42 Dr. Caton. It seems that Dr. Bates and Dr. Caton's position is that any increase in  
43 particulate matter, no matter how small, is unacceptable while a slight decrease is  
44 inconsequential – this seems inconsistent.  
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3 **Short-Term Health Effects**  
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5 **Q. Mr. Lepage testified at length about the possibility that short-term emissions**  
6 **associated with shut-down and start-up might be higher. Did he testify that**  
7 **there would be any adverse health effects associated with the higher emissions he**  
8 **claimed would occur?**  
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13 A. No. His testimony focused solely on the emissions volume, and I believe Mr. Hansen  
14 will address those claims. Mr. Lepage never claimed that any adverse health effect  
15 would result from these emissions.  
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21 **Q. Does Mr. Lepage's testimony about start-up and shut-down emissions raise any**  
22 **health concerns in your mind?**  
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25 A. No. Eric Hansen has provided information that indicates the emissions from start-up  
26 and shut-down would not significantly alter the maximum emissions reported earlier,  
27 in part because the start-up and shut-down events would be associated with periods of  
28 zero emissions. Therefore, based on testimony from Eric Hansen, start-up and shut-  
29 down will not have significant effects on the ambient concentrations of pollutants  
30 attributable to the proposed facility.  
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39 **Q. In her prefiled testimony, Jane Koenig expressed a concern about PM<sub>2.5</sub>**  
40 **emissions. How do you respond to that concern?**  
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43 A. Dr. Koenig expressed concern that there may be adverse health effects if the  
44 maximum SE2 emissions occurred on the same day as maximum background PM<sub>2.5</sub>  
45 concentrations in the Lower Fraser Valley. However, there are a few problems with  
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1 Dr. Koenig's suggestion. First, use of air concentration estimates derived from  
2 statistical representations (e.g., percentiles) has more scientific rigor than the use of  
3 single data points (such as the maximum). For example, in deriving the Canada-wide  
4 Standards for PM<sub>2.5</sub>, the Canadian Council of Ministers of the Environment (CCME)  
5 has indicated that standards based on the 98th percentile (averaged over 3 years) are  
6 more appropriate data to protect the health of the Canadian public.  
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14 Further to the above, data gathered by Eric Hansen from Canadian sources indicates  
15 that the maximum background value based on the metric proposed by the Canada-  
16 wide Standards is 18 ug/m<sup>3</sup> for the years for which there are sufficient data. A very  
17 conservative assessment of the cumulative PM<sub>2.5</sub> concentrations would add the 3.7  
18 ug/m<sup>3</sup> maximum impact anticipated from SE2 to the 18 ug/m<sup>3</sup> background value, for  
19 a total of 21.7 ug/m<sup>3</sup>, which is less than the levels noted by Dr. Koenig to be of  
20 concern (i.e., 25 ug/m<sup>3</sup> and 30 ug/m<sup>3</sup>). In referring to the Canada-wide Standard, it  
21 was also concluded by the Joint Technical Report (September 11, 2000) that "[w]hen  
22 the maximum predicted impact of PM<sub>2.5</sub> due to the facility emissions are added to this  
23 level it is unlikely that the proposed facility emissions will result in exceedances of  
24 this new standard" (page viii).  
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38 Finally, I should point out that compared to the absolute maximum background  
39 concentrations ever measured in the airshed, the impact of the SE2 facility becomes  
40 even more insignificant. To the extent that people are concerned about the health  
41 effects of ambient particulate matter concentrations in the Fraser Valley, the focus on  
42 the very slight contribution that would come from SE2 is misplaced.  
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**Regulatory Standards and Effects Thresholds**

**Q. In his pre-filed testimony, Robert Caton criticizes you for "re-hashing" the debate about whether adverse health effects occur even when regulatory standards are met. How do you respond to that criticism?**

A. I think he misunderstands my testimony. My point was not – as Dr. Caton implies – to simplistically conclude that: "as long as regulatory standards are met there can never be any adverse health impact." Rather, I made several points:

First, the ambient air quality standards and objectives established by Canadian regulatory agencies are valuable tools in evaluating the health effects of ambient air pollutant concentrations. These standards/objectives have been established based upon analysis of available scientific data by committees of scientists responsible for establishing standards/objectives that are considered acceptable from a health perspective for the Canadian population. The people contributing their time and expertise in developing the Canada-wide Standards represent many of Canada's leading scientists from government, academia and the private sector. It is the typical and accepted scientific practice for professionals to begin their risk assessment by comparing the effects of a project on ambient air quality to these standards.

Second, the predicted ambient air concentrations of pollutants resulting from the modified SE2 project are extremely small. Nothing in the scientific literature indicates that these very small incremental increases in ambient concentrations have resulted in a measurable impact to human health. I have conducted an extensive

1 review of the current scientific literature on PM<sub>10</sub>, PM<sub>2.5</sub> and ozone, and I have not  
2 found any study that showed health effects in epidemiological, animal toxicology or  
3 controlled human studies at the low levels predicted from the SE2 facility.  
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9 Conclusions about health effects from slight incremental increases in PM<sub>2.5</sub> or PM<sub>10</sub>  
10 are for the most part based on statistical associations using epidemiological data that  
11 reported effects associated with increases in 10 ug/m<sup>3</sup> or more PM<sub>2.5</sub> or PM<sub>10</sub>.  
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14 Statisticians use models with no threshold for health effects associated with exposure  
15 to particulate matter because epidemiological studies have been unable to demonstrate  
16 a threshold for ambient particulate matter. Empirical studies, however, have not  
17 shown measurable effects at the concentrations attributable to SE2 (i.e., maximum  
18 short-term impact in Canada 3.7 ug/m<sup>3</sup>).  
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26 Likewise, it is highly speculative to make conclusions about health effects from slight  
27 incremental increases in ozone based on statistical associations using data that report  
28 effects associated with increases of 20 ppb or more. The increases reported in the  
29 scientific literature quoted by Dr. Bates are about 100 times higher than the 0.2 ppb  
30 ozone impacts in Abbotsford that Environment Canada modeled based on the original  
31 project assumptions. Being able to calculate a theoretical increase in risk is very  
32 different than identifying a measurable impact in the real world. One needs to be  
33 extremely careful in according weight to small differences in calculated theoretical  
34 risks because the models for calculating these risks employ numerous assumptions  
35 and uncertainty factors.  
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1 So you see, my point is not that ambient levels of pollution below regulatory  
2 standards can never be harmful. My point is that there is no scientific reason to  
3 believe that the small increases in ambient air concentrations of pollutants resulting  
4 from the modified SE2 project will result in a discernible adverse impact on public  
5 health.  
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12 **Q. In his prefiled testimony, Dr. Bates refers to a recent study conducted during the**  
13 **1996 Summer Olympics in Atlanta that he claims demonstrates that health**  
14 **effects can occur when ozone levels below U.S. and Canadian standards. How**  
15 **do you respond to this study?**  
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21 A. There are several things worth noting.  
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25 First, as I explained, I have never contended that air pollution concentrations below  
26 established standards never result in health effects. Rather, I've pointed to the  
27 established standards as one important consideration in evaluating ambient air  
28 concentrations, and as a relevant consideration in determining "acceptable risk".  
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35 Second, the Atlanta study involved measurement of health impacts during an  
36 approximately 20 ppb decrease in ambient ozone concentrations. The modeling  
37 conducted by Environment Canada for SE2 (which assumed higher NO<sub>x</sub> emissions as  
38 well as a shorter emission stack) indicated that the maximum increase of ground level  
39 ozone concentrations would be less than 2 ppb within 5 km of the facility and less  
40 than 0.5 ppb more than 5 km away. Environment Canada concluded that ozone levels  
41 in Abbotsford would rarely increase more than 0.2 ppb as a result of the SE2  
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1 emissions. The Atlanta study (that evaluates ozone level changes approximately 100  
2 times greater than that estimated for Abbotsford) does not provide any evidence to  
3 dispute my opinion that the SE2 emissions will not result in a discernible impact on  
4 public health.  
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10 The Atlanta study cited by Dr. Bates indicates that measurable health effects can be  
11 found when there are large changes in ozone. However, this study does not provide  
12 evidence of measurable adverse health effects at the much smaller concentrations  
13 conservatively predicted for Abbotsford from the SE2 facility. Moreover, I note that  
14 Dr. Bates did not reference any scientific literature that provides evidence that the  
15 levels of ozone associated with the SE2 facility (i.e., 0.2 ppb ozone) would cause any  
16 health effects. This is not surprising since my review of the scientific literature  
17 indicates that no such information exists.  
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28 To provide more information regarding the above comments, in the paper cited by Dr.  
29 Bates, Friedman et al. (2001) found that the number of asthma acute care events  
30 decreased by 11.1 to 44.1% in various databases during the 1996 Summer Olympic  
31 Games in Atlanta when automobile use was markedly decreased. The authors  
32 reported that the peak daily ozone concentrations dropped by 27.9% from 81.3 ppb to  
33 58.6 ppb during the Olympic Games. In other words, an incremental change of 22.7  
34 ppb ozone was associated with a measurable impact on health. The greatest  
35 association with ozone the authors detected was in a health maintenance organization  
36 database whereby there had been 1.36 daily asthma acute care events during baseline  
37 conditions but only 0.76 daily events during the Olympic Games. Using this data for  
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1 illustration, if we assume a strictly linear relationship between ozone concentrations  
2 and asthma acute care events, a 0.2 ppb difference in ozone concentrations would  
3 translate to a calculated difference of 0.005 daily asthma acute care events (a change  
4 from 1.36 to 1.355) in that particular database as a worst-case estimate. From my  
5 perspective, this would not be a measurable impact in the real world. Indeed, the  
6 difference could easily fall within the "margin of error" and the uncertainties inherent  
7 in the analysis.  
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17 The problem that I see with Dr. Bates' position is that it suggests that any increase in  
18 air pollution will have adverse health effects and will be unacceptable. It seems to  
19 ignore the concept of *de minimis* risk that plays a necessary role in the risk assessment  
20 process.  
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27 **Q. Can you further explain the concept of *de minimis* risk?**

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29 A. *De minimis* risk is a concept that forms the basis of much of the regulatory policy in  
30 Canada and the United States. It essentially defines a level of risk beyond which it is  
31 not considered to be significant for further regulation. This allows regulatory agencies  
32 to focus on issues of greatest concern while not expending efforts on issues that will  
33 have little or no impact on the health of the general public.  
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41 **Q. Dr. Caton testified that "Any amount of additional pollution increases the risk of  
42 occurrence of many respiratory and cardiac diseases. There has been no  
43 showing of thresholds below which impacts do not occur." Likewise, Dr. Bates  
44 testified that "there is no safe threshold below which these pollutants do not  
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1 **cause health problems and that, as these pollution levels increase, so does the**  
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3 **risk of adverse health effects." How do you respond to their arguments?**  
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5 A. First of all, I think it is important that we understand that Dr. Caton and Dr. Bates are  
6 talking about risk. We all face all kinds of risks everyday and many of those risks  
7 have no known safe threshold as Dr. Caton puts it. For example, we risk breaking our  
8 neck every time we step into the bathtub to take a shower. There is no absolutely safe  
9 threshold – we could slip the first time we try it – and, in theory, the risk increases the  
10 more times we do it. At the risk levels we're talking about, however, the difference  
11 between people showering once a day versus once a week probably doesn't result in  
12 any discernible effect on public health, and I certainly don't know anyone advocating  
13 that we only shower once a week in order to reduce the very small public health risk it  
14 poses.  
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26 Admittedly, that's obviously a silly example in some sense, but it does remind us that  
27 generalities about "risks being linear" and "no safe thresholds" aren't always  
28 particularly helpful. As Dr. Caton acknowledges, "[t]he real issue is one of  
29 acceptable risk." My point is that the air pollution related health effects associated  
30 with existing ambient air quality near Sumas and those associated with the ambient  
31 air quality predicted once SE2 is operating are indiscernible in practice. It is always  
32 possible to calculate a theoretical difference in risk if you make enough assumptions,  
33 accept enough uncertainties in your risk statistics and use enough significant figures,  
34 but that doesn't make the calculated difference relevant to regulatory decision making.  
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1 **Q: Dr. Bates suggested that your testimony implies that a threshold air**  
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3 **concentration has been identified for particulate matter. Did you state this?**

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5 A. No. One of his main concerns regarding my testimony seems to surround a  
6  
7 misinterpretation of my discussion on threshold concentrations for health effects from  
8  
9 particulate matter. Dr. Bates concluded that I contend that there is a threshold for  
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11 health effects associated with exposure to ambient particulate matter; however, my  
12  
13 testimony is clear that a threshold concentration for absolute protection of health  
14  
15 effects has not yet been identified. Nevertheless, it is still possible to develop air  
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17 concentrations for particulate matter that would be protective of the general  
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19 population and associated with acceptable risks. Let me expand on this.

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23 There are two types of substances that are commonly evaluated by regulatory  
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25 agencies: (a) threshold response substances; and (b) non-threshold response  
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27 substances. A threshold response substance will have an air concentration for which  
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29 no known health effects are expected in the general population. Development of an  
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31 acceptable air concentration for a threshold response substance usually involves  
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33 evaluation of a wealth of data that leads to a scientific consensus that virtually no  
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35 members of the general public would be affected. When the air concentration of such  
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37 a substance is less than the threshold concentration, no appreciable risk of adverse  
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39 health effects would generally be expected. Threshold air concentrations have been  
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41 developed for various chemicals for a number of substances whose mechanisms of  
42  
43 toxicity are well understood.

1 There is also a group of substances known as the non-threshold response substances.  
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3 For these substances, air concentrations cannot be determined that are associated with  
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5 zero risk and it is generally assumed that even one molecule (or particle) of the  
6  
7 substance may have a certain level of theoretical risk to human health. As a result,  
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9 these substances are regulated based on the concept of “acceptable level of risk”  
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11 whereby risk assessment specialists, medical professionals and other scientists  
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13 propose an air concentration of a substance that would have an acceptable level of  
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15 risk to the general public. Acceptable levels of air concentrations of non-threshold  
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17 response chemicals have been developed for many substances commonly found in air.  
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19 Without the concept of acceptable risk for non-threshold substances, activities such as  
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21 starting your car, cooking in your house, or having a campfire would not be possible  
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23 since all of these activities would be associated with some contribution of risk from  
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25 exposure to substances.  
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28 To date, a clear threshold air concentration whereby absolute protection of the general  
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30 public would be ensured has not been identified for particulate matter. However,  
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32 laboratory and epidemiological studies report measurable health effects only when  
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34 particulate matter is increased in increments much greater than would result from the  
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36 proposed SE2 facility.  
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40 Regulatory agencies have proposed air concentrations that are considered to be  
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42 sufficiently protective of human health effects even if there is no threshold  
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44 concentration for effects from particulate matter. As stated in my prefiled testimony,  
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46 the most relevant guidance for evaluating health effects in Canada from the proposed  
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1 SE2 facility are the Canada-wide Standards and the BC Environment Objectives that  
2 were established for protection of the health of the general public and the  
3 environment. These standards minimize human health risks to levels that Canadian  
4 regulatory agencies deem acceptable.  
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11 **Q. In support of his point that increases in pollution "translate to increases in**  
12 **adverse health effects," Dr. Bates points to a study involving Los Angeles school**  
13 **children and another study conducted in Boston. How do you respond to these**  
14 **studies?**  
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19 A. First of all, I must point out again, that I am not contending that there is an absolutely  
20 safe threshold of pollution, and I am not disputing that risks generally increase as  
21 pollution increases. My point is that the extremely small changes in ambient air  
22 concentrations of pollutants as a result of the SE2 project will not result in a  
23 measurable effect on human health.  
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31 The Los Angeles study Dr. Bates cites does not demonstrate a discernible health  
32 effect from the small increases in ozone levels associated with SE2. Similar to the  
33 Atlanta study, the Los Angeles study identified health impacts with a 20 ppb increase  
34 in ground-level ozone concentrations. That's about 100 times the concentration  
35 attributable to SE2 in Abbotsford. Therefore, the health effects associated with ozone  
36 exposure two orders of magnitude higher than that projected for Abbotsford are not  
37 directly comparable.  
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1 The Boston study concerns particulate matter and again it fails to demonstrate a  
2 discernible health effect from the very small increases in particulate levels associated  
3 with the SE2 project. The Boston study evaluated “[w]hether high concentrations of  
4 ambient particles can trigger the onset of acute myocardial infarction” associated with  
5 ambient air concentration increases of 20 to 25 ug/m<sup>3</sup> PM<sub>2.5</sub> over 2 hours or 24 hours.  
6 This increase in PM is much greater than that predicted from the SE2 facility, which  
7 averages 0.38 ug/m<sup>3</sup> and has a maximum of 3.7 ug/m<sup>3</sup>. The article is similar to  
8 previous literature in this field, as Dr. Bates indicates, and does not alter any of the  
9 conclusions made in the prefiled testimony. None of the literature available since the  
10 Canada-wide Standards were implemented would change any previous conclusions  
11 that have been made. My final assessment of the potential for health effects  
12 associated with exposures from the SE2 facility take into account the volumes of  
13 literature that are available on this topic.  
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29 Put simply, to my knowledge, no empirical study has ever demonstrated a statistically  
30 significant change in health impacts associated with such a small change in ambient  
31 air concentrations.  
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37 **Q. Dr. Caton states that "health risks are directly correlated to the project's**  
38 **emissions and . . . the project changes will result in very little change in the**  
39 **overall emission rates." How do you respond to that statement?**  
40

41  
42 **A.** Health risks are not directly correlated with project emissions. As I explained in my  
43 direct testimony, the key issue is not the amount of emissions but rather the resulting  
44 ambient air concentrations of the pollutants at issue. As Eric Hansen explained in his  
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47

1 testimony, SE2 made several changes to the project that resulted in reducing potential  
2 effects on ambient air quality. Some of those changes involved lowering emissions,  
3 but another – increasing the stack height – lowered ambient air concentrations by  
4 increased the dispersion of emissions. I've already discussed the changes in ambient  
5 air concentrations attributable to the revised project, and my reasons for concluding  
6 that the revised project will not result in a measurable effect on public health.  
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14 **Q. Dr. Bates expressed concern regarding the increases in sulfuric acid mist and**  
15 **SO<sub>2</sub> might result in adverse health effects. Do you agree with him?**  
16

17 **A.** No. While the levels of SO<sub>2</sub> are higher than in the original application, the levels are  
18 much lower than background concentrations, and the additive SE2 emissions with  
19 background concentrations are still an order of magnitude lower than the most  
20 stringent Canadian objective for SO<sub>2</sub> for any of the time frames modeled. Similarly,  
21 the maximum levels of sulfuric acid mist were estimated to be up to 0.29 ug/m<sup>3</sup>,  
22 which is very low. I am not aware of any scientific literature that has identified  
23 potential health effects to be associated with the modeled levels of SO<sub>2</sub> and sulfuric  
24 acid aerosols.  
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37 **Q. Dr. Bates and Dr. Caton seem to imply that health impacts from the proposed**  
38 **SE2 facility will be great. Do you agree with them?**  
39

40 **A.** No. I know of no study that has demonstrated measurable health effects from the  
41 incremental increases estimated to be associated with the proposed SE2 facility. I  
42 acknowledge that if you make sufficient assumptions, it may be possible to calculate a  
43 slightly higher theoretical risk associated with the slightly higher concentrations of  
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1 particulate matter and ground-level ozone associated with the revised project;  
2  
3 however, this does not mean that the slight increase in theoretical risk is unacceptable  
4  
5 or that it will result in measurable effects on public health. Using the rationale  
6  
7 proposed by Dr. Bates and Dr. Caton, one might also argue that any number of  
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9 activities, such as cooking, starting a car, etc., should also not be permitted since these  
10  
11 may result in increased theoretical risks.  
12  
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14  
15 A pragmatic person would agree that there are some incremental increases which are  
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17 indistinguishable (or *de minimis*) from that posed by existing background conditions.  
18  
19 At this time, little published information is available on what should be considered to  
20  
21 be a *de minimis* increase in particulate matter concentrations. However, information  
22  
23 presented in my earlier testimony may partially shed light on this issue. BC Ministry  
24  
25 of Environment, Lands and Parks (1995) states that a 1 ug/m<sup>3</sup> increase in PM<sub>10</sub> is “a  
26  
27 change that everyone likely would agree is insignificant, regardless of the estimated  
28  
29 impacts of such a small change” (p. 48, “Health Effects of Inhalable Particles:  
30  
31 Implications for British Columbia: Overview and Conclusions”, June 1995). Annual  
32  
33 increases in ambient concentrations of particulate matter resulting from the revised  
34  
35 SE2 project would be only a fraction of this level.  
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38  
39 Overall, I consider that increases in particulate matter of the magnitude estimated for  
40  
41 the proposed SE2 facility are acceptable or *de minimis* from a human health  
42  
43 perspective.  
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1 **Q. The Province's witnesses also appear to contend that the linear relationship**  
2 **between pollution and health risks provides a strong argument for not allowing**  
3 **the SE2 project to be built near the Fraser Valley airshed. Do you agree?**  
4

5  
6  
7 A. It seems inconsistent that a witness who contends that any increase in pollutants  
8 would result in adverse health effects would suggest that a facility should be located  
9 in another location. On the contrary, according to those witnesses, any incremental  
10 increase in ambient concentrations of pollution is associated with an increase in risk,  
11 which presumably means that the same increased individual risk would be present  
12 wherever this sort of power project were located. The argument then becomes nothing  
13 more than a "not in my backyard" argument. As I've explained previously, I do not  
14 believe that slight increases in ambient concentrations of particulate matter and ozone  
15 resulting from the SE2 facility will result in measurable impacts on public health.  
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27 **Q. Dr. Bates and Dr. Caton imply that you are being inconsistent with your support**  
28 **of SE2's offset proposal. How do you respond to them?**  
29

30  
31 A. Based on my concern for air quality, I strongly support the use of best available  
32 control technology (BACT) wherever possible, including mobile sources, which have  
33 been identified as a major source affecting ambient air quality. From my perspective,  
34 voluntary commitments to provide offsets plans for other sources could reduce  
35 ambient air concentrations of some pollutants, which could potentially result in a  
36 measurable improvement in public health. I do not think that supporting these  
37 voluntary commitments compromises my earlier position that the SE2 facility  
38 emissions will not pose an unacceptable health risks.  
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**END OF TESTIMONY**