

Preliminary Comments on the ISA from Dr. Richard Schlesinger

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4 P. 4.4, L. 16-18. Bronchoconstriction can be initiated by irritants in the URT as well as
5 those in the LRT
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7 p. 4.8, l. 27. Proportion should read proportional
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9 p.4-10, l. 18-20. It is not clear what is meant by even during exercise since with exercise,
10 there is a switch to even increased mouth breathing
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12 p.4-10, l.26-27 It states that the rate and route of breathing both have great effects on the
13 magnitude of SO₂ absorption in the URT and penetration of SO₂ to the
14 lower airways. However, earlier in the chapter it is stated that, “during
15 normal breathing...95% or greater SO₂ absorption occurs in nasal
16 passages...”. These two comments seem to contradict each other.
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18 p.4-24, l 17-18 It is not clear how the cited study supports the statement since it was a
19 combination of both SO₂ and NO₂
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21 p.4-27 l.35 -4.28, l. 1-7 The last sentence indicating that the study provides consistency with
22 SO₂ playing a role in the exacerbation of AHR is an overreach, since as
23 noted, the other components of the atmosphere may have contributed to
24 the response. This study should not be cited here as evidence for
25 exacerbation of AHR.
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27 FIG 4-2 Short term effects are ascribed solely to the formation of sulfite in the ELF
28 and the potential effect of direct irritant effect due to formation of H⁺ is
29 ignored in this paradigm
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31 FIG 4-3 This suggests that similar outcomes and endpoints occur by different key
32 events in long term vs short term exposures. Is that actually the case? The
33 text notes that the effect of long term exposures is due to the formation of
34 reactive products in ELF, which are the same ones following short term
35 exposure. This is confusing.
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