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Author's Response

Sir,

Mr. Wigmore raises several points related to the measurement of blood–breath ratio (BBR). Indeed, error at low breath alcohol concentrations (BrAC) and blood alcohol concentrations (BAC) can play a proportionally larger role than would be the case at higher BAC levels. However, these error issues do not help with the understanding of the new paradigm (1) that was developed to explain anomalies in the old paradigm found by several scientists: (i) near-zero dead space (initial exhaled volume with no alcohol) (2); (ii) no alveolar plateau while exhalation continues (2); (iii) BrAC increases with increasing exhaled volume (2); (iv) BrAC depends on prebreath test breathing pattern (3,4); (v) directly measured alcohol blood/air partition ratio at body temperature is 1783 ± 8.1 and 1830 ± 7.8 for men and women, respectively rather than 2100 (5); (vi) warmed (isothermal) rebreathing BBR varies between 1947 ± 110 (6) and 2019 ± 121 (4) rather than 2100. The new paradigm provides a model to explain these findings that cannot be explained by the old paradigm.

The concept of deposition of alcohol on the airway surface during exhalation was developed in the late 1960s and early 1970s by Wright et al. (7) and was briefly mentioned in one sentence by Begg et al. (8). Even though this deposition mechanism was introduced more than 40 years ago, little attention has been paid to the implications of this airway deposition. If conditions exist for deposition during exhalation, then the conditions also exist for the uptake of alcohol from the airways during inhalation.

Mr. Wigmore lists a number of experimental errors than can contribute to variability in the measurement of BBR. Certainly, the factors mentioned all contribute to uncertainty in the BBR. However, under the new paradigm, which recognizes airway alcohol exchange during both inhalation and exhalation, the alcohol exchange does not occur in the alveolus. Therefore, considering a ratio such as blood to breath no longer applies to the alcohol breath test. The breath alcohol concentration is related to arterial blood concentration and a number of physiological factors that are not controlled, such as prebreath test breathing pattern, inhaled air volume, exhaled breath volume, and lung volume. Such factors dominate the determination of breath alcohol concentration.

The new paradigm recognizes the uptake of alcohol from the airways during inhalation as well as the redeposition of alcohol on the airways during exhalation (see figure 4 of the Paradigm Shift paper [1]). The net result is that the alcohol concentration in each alveolus is approximately 15–20% greater than the average alcohol concentration at the mouth (9). The notion that the end-exhaled alcohol concentration is similar to the alveolar alcohol concentration (an essential tenet of the old alcohol breath test model) is not true. The difference between exhaled alcohol concentration and alveolar alcohol concentration is governed by variables such as the volume of air inhaled prior to the breath test, the volume of air exhaled into the breath test machine, and the duration of breath hold prior to exhaling into the breath test machine. Because such factors are not controlled when the breath alcohol test is administered, there are uncontrolled error factors that are not accounted for by the old paradigm.

The new paradigm predicts that the alcohol exchanges almost entirely within the airways, implying that little or no alcohol exchanges in the alveolus. This is a dynamic process depending on the ventilation rate during both inhalation and exhalation. Henry's law (requiring equilibrium conditions) no longer applies to the alcohol breath test because alcohol does not exchange in the alveolus.

The bronchial arterial blood (coming from the left heart) brings alcohol to the airways of the lungs. Alcohol reaching the mouth comes from the airways and the systemic arterial blood system. This is part of the new paradigm and consistent with the observations of Lindberg et al. (10) who found that the breath alcohol concentration (obtained with an instrument using free exhalation rather than blowing through a tube, adding resistance) correlated more closely with arterial blood rather than venous blood during both the absorptive and postabsorptive phase. In that study, the authors corrected the measured exhaled alcohol concentrations back to what it would have been in the alveolus by adjusting alcohol in proportion to the decrease in water vapor concentration. Their correction assumes that no water vapor or alcohol exchange occurs in the airways. Appropriate adjustment would require correction for both the airway alcohol exchange as well as the dilution of exhaled breath by entrained room air. Correction of exhaled alcohol concentration to that in the alveolus was made by correcting the alcohol concentration by the dilution of water by entrained room air to the presumed water content in the alveolus while ignoring the differential airway exchange of both alcohol and water vapor. Because the authors did not correct for airway exchange of both alcohol and water vapor, the partition ratios calculated by Lindberg et al. (10) would not have reflected the effects of lung volume on airway size (the source of the possible lung volume–dependent bias). Our lung model (11) predicts that a person with a lung volume of 5.0 L will lose around 6.1% of water vapor and 21.7% of alcohol by the time the air passes from the alveolus to the mouth. A person with a lung volume of 4.0 L will lose 6.2% of water vapor and 21.1% of alcohol with a full inhalation from functional residual volume to total lung capacity and a full exhalation to residual volume. The loss of alcohol to the airway surface is greater than the loss of water, and the loss of alcohol depends on lung airway size and, of course, other breathing parameters. Therefore, neglecting airway exchange will hide the lung volume dependence of the alcohol breath test. This may be the explanation for Lindberg et al. finding similar BBR for both men and women. Certainly, more data are needed regarding the potential bias of lung volume in the alcohol breath test.

Jones and Andersson (12) found that average female BBR exceeded average male BBR by 5.6%. Their results were not statistically significant because they had to use an unpaired *t*-test and the variation among the male and female subjects was greater than the mean difference. Significance could not be obtained because lung volume was not measured in their subjects. Our computer model predicted a similar difference between men and women based on changes in airway dimensions with changes in lung volume. Skåle et al. (13), in their abstracted article, found a positive correlation between body weight and BBR. Subjects with greater body weight usually have greater lung volume (but not at the extremes). While neither of the above-mentioned studies measured lung volume, their findings are both consistent with a lung volume dependence of BBR. Future researchers studying the alcohol breath test should consider measuring vital capacity for each subject.

Mr. Wigmore refers to an "arterial-venous lag" in alcohol concentration. During the absorptive phase, arterial concentration exceeds venous alcohol concentration as the absorbed alcohol

passes from the intestines to the heart and the systemic arterial blood. During the postabsorptive phase, venous alcohol concentration exceeds arterial blood alcohol concentration. While this difference might be considered a "lag," it is simply a difference to reflect whether alcohol is being delivered to the peripheral tissue (during absorption phase) or being taken up from the peripheral tissue (during postabsorption phase). The strong correlation of arterial blood concentration with breath alcohol concentration during both the absorptive and postabsorptive phase is consistent with the new paradigm in that alcohol exchanges with the airway surface and the bronchial circulation. Because of the new paradigm, future blood–breath correlation studies should be performed using arterial blood. The past venous blood and breath correlation studies are therefore neither probative nor helpful for understanding mechanisms of alcohol exchange.

Up to the present, the forensic community has steadfastly presumed that end-exhaled alcohol concentration is strongly related to alveolar alcohol concentration, which is directly related to venous blood alcohol concentration via Henry's law without direct experimental confirmation. Evidence is mounting that breath alcohol is more closely related to arterial blood because the exchange occurs in the airways, not the alveolus. Henry's law no longer applies.

It is time to consider the new paradigm in research studies within the forensic alcohol community. We should begin on a pathway toward either improving the alcohol breath test by controlling the relevant variables or abandoning the alcohol breath test altogether.

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