# Authors' reply

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## Dear editor

Dr Motwani states that the purpose of his paper is to "demonstrate how HOAs can cancel out modify or induce lower order corneal astigmatism."[1] He hypothesizes that anterior corneal higher-order aberrations (CHOAs) and anterior corneal astigmatism (ACA) interact, and for illustrative purposes he describes them as creating 2D ovalizations in the central cornea, "depending on how the two ovals of higher-order and lower order line up, it will either increase or decrease the manifest measurement of astigmatism."[1] He acknowledges that his simplification is "attempting to break down complex three-dimensional interactions between HOA and lower order astigmatism."[1] He mentions the possibility of specifically quantifying the axes of the ovals and "creating a vector diagram that can accurately predict the axis."[1] We did precisely that.[2] To objectively investigate his theory based on approximations, we developed an advanced algorithm that analyzed the Contoura ablation profile in 3D, accurately detailing the CHOAs ellipse.[2] We then studied the vectorial relationship between the calculated CHOAs and ACA ellipses. Once mathematically quantified, our data shows that his notion of ovals interacting does not hold true for 50% of eyes.[2] He also gives an example of a case that is not explained by his theory. Dr Motwani's response that CHOAs are undermeasured because of epithelial compensation is another interesting topic to explore but cannot explain our empirical analysis that does not support his theory.[1] His logic that the ovalization hypothesis is valid and that ocular residual astigmatism (ORA) is caused mainly by CHOAs – because treating on the ACA has "not worsened patients' vision" – is not scientific.[1] Furthermore, his Part 3 paper (which is not the subject of our study) only presents 50 eyes, no control group, with a small mean RA to ACA magnitude discrepancy of 0.55 D (not high ORA), and does not include the standard reporting refractive surgery outcome graphs, nor astigmatism vector analyses. One cannot make definitive conclusions of accuracy and "astigmatism elimination,"[1] nor make a recommendation for treating solely the ACA, especially in high ORA cases, based on such preliminary data and incomplete analysis.

Dr Motwani claims that posterior corneal astigmatism (PCA) does not contribute significantly to RA.[1] That claim negates many previous studies that are not referenced in his paper,[3–5] and his observations from clinical practice that few ACA treated eyes have postoperative astigmatism has not been substantiated with rigorous outcomes data. Dr Motwani misunderstood our term "additive" as meaning PCA increases RA, but it refers to vectoral addition in which the cornea is subtractive most of the time. Published data shows that 87% of posterior corneas produce against-the-rule (ATR) refractive astigmatism by acting as a negative lens.[4] This finding, with some contribution from CHOAs and cerebral preference for with-the-rule (WTR), likely contributes to the decreased subjective RA seen in most WTR corneas, and the increased subjective RA in most ATR corneas, which we described in 5,403 eyes.[2] PCA magnitude and its impact is frequently small, but in certain cases it can be significant and therefore cannot just be ignored. There is a complex interplay of compensatory mechanisms optimizing subjective vision. Postoperatively, these include biomechanical corneal shifting, epithelial remodeling, internal compensation of induced CHOAs, and as Dr Motwani describes, cerebral processing. Rigorous studies of thousands of eyes are needed to truly understand the interaction of CHOAs, ACA, PCA, and the effect of a combined CHOA and lower-order astigmatism excimer ablation. We thank Dr Motwani for his comments and contribution to the discussion surrounding this important topic.

## Disclosure

The authors report no conflicts of interest in this communication.

## References

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