Table. Clinical characteristics and hospital course of infants

	Bokodi et al		Kazzi and Quasney	
	DD/DI (n = 105)	II (n = 9)	DD/DI (n = 97)	II (n = 23)
Birth weight (g)*	1233 ± 358§	1497 ± 472§	938 ± 204	925 ± 196
Gestational age (wk)*	30 ± 3	31 ± 4	28 ± 3	28 ± 2
Male†	51%	67%	42%	48%
Respiratory distress syndrome†	49%	56%	76%	74%
Days of mechanical ventilation‡	2 (0–8)	1 (0–7)	13 (1–45)	8 (1–27)
Days of oxygen supplementation#	3 (0–8)	11 (3–13)	52 (7–74)	31 (3–60)
BPD†	18 (17%)	3 (33%)	46 (47%)§	5 (22%)‖
No BPD†	87 (83%)	6 (67%)	51 (53%)§	18 (78%)‖

The parameters written in bold letters are different in the two studies.
*Student's *t* test. Values are mean ± SD.
†χ^2 test. Values are percentagees of infants.
‡Mann-Whitney U test, values are median (25–75th %).
§$P < .05$;
‖$P = .025$.

To the Editor:

We read with great interest the article of Kazzi and Quasney about the possible implication of the angiotensin-converting enzyme (ACE) DD/DI genotype in bronchopulmonary dysplasia (BPD).[1] Our team also investigates the association of genetic polymorphisms with perinatal morbidity. In addition to several cytokine gene polymorphisms,[2,3] we also determined ACE D/I and AT1R A1166C polymorphisms in more than 100 babies born with a birth weight ≤1500 g (Table).[4,5] Based on the idea suggested by Kazzi and Quasney, we re-analyzed our data and tested whether the ACE DD/DI genotype presents an increased risk for BPD in our population. Interestingly, we did not detect any association between ACE and AT1R genotype and BPD or ventilation characteristics.

The discrepancy between the population enrolled in our study and that of Kazzi and Quasney may be the probable explanation for the different results (Table). Our population was more mature; required less ventilation support; and the prevalence of BPD was also lower. Therefore, we think that the observation done by Kazzi and Quasney may be limited to a very immature population with a birth weight <1250 g. The marked alteration of the functionality of the renin-angiotensin system during fetal maturation[6] is a phenomenon supported by several studies. Perhaps the impact of ACE genotype on lung function is the more pronounced during the earlier phase of fetal life and decreases in more mature preterm infants.

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REFERENCES

1. Kazzi SN, Quasney MW. Deletion allele of angiotensin-converting enzyme is associated with increased risk and severity of bronchopulmonary dysplasia. J Pediatr 2005;147:818-22.
2. Treszl A, Kocsis I, Szathmári M, Schuler Á, Héninger E, Tulassay T, et al. Genetic variants of TNF-α, IL-1β, IL-4 receptor α-chain, IL-6 and IL-10 genes are not risk factors for sepsis in low birth weight infants. Biol Neonate 2003;83:241-5.
3. Treszl A, Héninger E, Kálmán A, Schuler Á, Tulassay T, Vásárhelyi B: Lower prevalence of IL-4 receptor alpha-chain gene 1902G variant in very low birth weight infants with necrotizing enterocolitis. J Ped Surg 2003;83: 1374-8.
4. Nobilis A, Kocsis I, Tóth-Heyn P, Treszl A, Schuler A, Tulassay T, et al. Variance of ACE and AT1 receptor gene do not influence the risk of neonatal acute renal failure. Ped Nephrol 2001;16:1063-66.
5. Treszl A, Szabó M, Dunai Gy, Nobilis A, Machay T, Tulassay T, et al. Angiotensin II type 1 receptor A1166C polymorphism and prophylactic indomethacin treatment induced ductus arteriosus (DA) closure in very low birth weight neonates. Ped Res 2003;54:753-55.
6. Norwood VF, Fernandez LG, Tufro A, Gomez RA. Development of the renin-angiotensin system. In: Polin RA, Fox WW, Amban SH, eds. *Fetal and Neonatal Physiology*. 3rd ed. Philadelphia: Saunders;; 2004: 1249-55.

Reply

To the Editor:

We thank Dr Lin et al for their interest in our work and appreciate the opportunity to respond to their comments. Per their suggestion, we re-analyzed the distribution of genotypes of angiotensin converting enzyme (ACE) among our infants after including death or bronchopulmonary dysplasia (BPD) at 36 weeks postmenstrual age as our primary outcome variable. As shown in the Table, the DD/DI genotype remains predominant among infants who died or developed BPD compared with infants who survived without BPD.

As to their second comment, we did not perform a systematic collection of data on infants' fluid intake, weight loss, oxygen requirement at 24 hours of age, and target level of oxygen and carbon dioxide saturation as they