

PLSC 731: Paper Review, Association Mapping

Agrama et al (2007) Association mapping of yield and its components in rice cultivars. Mol Breeding 19:341

1. What is the traditional rice QTL mapping approach? (341-342)
2. What method of QTL mapping is used in human? Why? (342)
3. What type of variation in linkage disequilibrium (LD) distances is observed in plants? (342)
4. Why are SSRs good markers to detect population structure? (342)
5. What is the value of using breeding germplasm for association mapping studies? (342)
6. What is the relationship between the number of markers and LD? (343)
7. How can unlinked markers be in linkage disequilibrium? (343)
8. What is the common feature of the AM population used in this paper? Why is it important? (343)
9. What phenotypic data was collected? Is this appropriate data? (343)
10. How many SSRs were tested? Is this enough? (343)
11. What software was used in the statistical analysis? What analysis was performed with each software? (345)
12. Are the phenotypic trait data normally distributed? Or does the paper report distribution data? (346)
13. Were the SSR markers sufficiently polymorphic? (346)
14. Why does $k=3$ fit the expected structure of the rice population? (346)
15. How much “ancestry” of a single subpopulation was necessary to assign an individual to a subpopulation? Was this an appropriate value? (346)
16. What was the best k value for the STRUCTURE analysis? What unifying feature was observed within a subpopulation? (346)
17. How were the eight Euclidian distance clusters defined? (Hint: they were not) (348)
18. What degree of variation was observed within/between clusters? (348)
19. What does F_{ST} tell you about the level of differentiation? (348)
20. At what distance did LD start to decay? (348)
21. How many marker trait associations were observed? (348)
22. Were the AM results consistent with previous QTL mapping studies using biparental populations? (350)
23. How might population structure data be used for parent selection in a plant breeding program? (350)
24. What might explain significant LD between unlinked markers? (352)
25. Is 150 SSR markers sufficient for LD studies? (352)
26. What would explain high levels of LD in rice? (352)
27. What type of errors might arise if population structure is not accounted for? (352)
28. What is the difference between LD estimates in this study and others? How do the methods differ? (353)
29. Is it true that AM can detect genetic factors with weaker effects than with biparental QTL mapping? (353)