would lead to failure of suppression of HOXB-8 expression in the anterior limb field. Ectopic expression of HOXB-8 would then specify cells in the anterior limb field as ZPA, leading to partial or complete duplication of grampsus, autopod, and stylopod.

There are no reports of grampsus (humerus/femur) duplication in Laurin-Sandrow syndrome, which would argue against the above hypothesis. To my knowledge, however, no author has to date thoroughly examined the humeri and femora of Laurin-Sandrow patients for any evidence of abnormal patterning. Further delineation of the Laurin-Sandrow phenotype, especially with regard to more proximal skeletal elements, would be most helpful in the search for candidate genes for this intriguing syndrome.

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A study of brothers with Klinefelter syndrome

Klinefelter et al described males with infertility, hypogonadism, and gynaecomastia in 1942. The other clinical features of Klinefelter syndrome are now well delineated but not present in all affected subjects.1 The live birth prevalence of Klinefelter syndrome is approximately 1 in 1000.2 The diagnosis is made and confirmed by the cytogenetic finding of 47,XXY. Klinefelter syndrome is considered to be a sporadic disorder resulting from non-disjunction. This is maternally in one half of cases and paternally in the remainder.3 The empirical recurrence risk is negligible as the condition occurs sporadically, with the recurrence risk equal to the birth prevalence, that is, 1 in 1000.3 The empirical recurrence risk is negligible as the condition occurs sporadically, with the recurrence risk equal to the birth prevalence, that is, 1 in 1000.3 The empirical recurrence risk is negligible as the condition occurs sporadically, with the recurrence risk equal to the birth prevalence, that is, 1 in 1000.3 The empirical recurrence risk is negligible as the condition occurs sporadically, with the recurrence risk equal to the birth prevalence, that is, 1 in 1000.3 The empirical recurrence risk is negligible as the condition occurs sporadically, with the recurrence risk equal to the birth prevalence, that is, 1 in 1000.3

Figure 1 Pedigree of the males with Klinefelter syndrome shown as filled squares. Cytogenetic analyses and X chromosome inactivation patterns are shown for the nuclear family. The latter were generated using the microsatellite present in intron 13 of the factor VIIIIC gene. The results are given using the gross up reaction products measured in base pairs.

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