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Addendum

Since submitting this report, a relevant paper has been published describing 3 further cases of X-autosome translocation (Zabel, B. U., Baumgart, W. A., Pirntke, W., and Gerhard-Ratschow, K. (1978). X inactivation pattern in three cases of X-autosome translocation. American Journal of Medical Genetics, 1, 309–317). Two of these, a phenotypically normal mother and her abnormal child had an X;15 translocation. In the chromosomally balanced mother, the normal X was preferentially inactivated, but in the child the abnormal X was late replicating in 75% of the cells, whereas in the remainder of the cells the normal X was inactivated. The abnormalities were therefore ascribed to partial trisomy 15q. The third case showed an X;21 translocation in which the normal X was inactivated in 70% and the Xp chromosome in 30% of the cells, but the chromosome 21 bearing the translocated Xp was never inactivated. The abnormal phenotype was ascribed to possible disomy of Xp, as in our patient, and lends further support for the existence of only one inactivation centre located in the proximal part of Xq (Therman and Patau, 1974).