The degradation is in mammalian cells stimulated by adenomatous polyposis coli (APC) protein and the members of the axin family. Inhibition of the GSK activity (e.g. by activation of Wnt signaling) elevates the levels of extrajunctional (soluble) ß-catenin and leads to its nuclear translocation. ß-catenin associates with the members of the LEF/TCF family in the nucleus and this is sufficient to activate genes that are first of all influenced by the Wg/Wnt pathway. A likely candidate target gene in *Xenopus* is the homeobox gene *siamois* involved in specifying the dorsal axis (see Ben Ze'ev and Geiger, 1998, for a rewiev). Very recent results by Tetsu and McCormick (1999) on ß-catenin-cyclin D1 expression positive coupling indicate that variations in the level of ß-catenin might be also directly responsible for the cell growth rate and cell proliferation.

In *Xenopus* development, however, APC can function to activate Wnt signaling and induce axis duplication (Vleminckx et al., 1997), a process normally dependent on increased ß-catenin levels. ß-catenin null-mutations result in very early (gastrulation) defects in the mouse embryo (Haegel et al., 1995), while plakoglobin-knockout embryos progress normally through early stages of development, but die later as a result of failure in heart development (Bierkamp et al., 1996; Ruiz et al., 1996). No information is available about the possibility of direct or indirect coupling between the level of ß-catenin and expression of other catenins or cadherins. The data of Reynolds et al. (1994) reflect no changes in the physical interactions of cadherin-associated proteins under the conditions in which they were phosphorylated on tyrosine. The levels of each cadherin-associated protein in the complexes from normal and transformed cells were very similar. No phosphorylation-dependent degradation of cadherins or catenins was reported. Hamaguchi et al. (1993) has also found apparently normal N-cadherin complexes in Src-transformed fibroblasts, and these complexes contained tyrosine-phosphorylated catenins. In another study, the phosphorylation of ß-catenin by epidermal growth factor receptors was shown to result in detachment of the entire cadherin-catenin complex from the cytoskeleton (Hoschuetsky et al., 1994). It is very likely that Src-induced phosphorylation of cadherin-associated proteins also leads to a similar effect (Reynolds et al., 1994; Cowin and Burke, 1996).

As shown here cadherins and catenins were virtually not detectable in the tissues of strongly aberrant B_1 frog embryos. At the same time, the levels of c-Src and phosphotyrosine staining increased. The mechanism of the disappearance of cadherins and catenins in the aberrant embryos has not been understood yet. A higher level of phosphotyrosine staining and a slightly higher Src staining were also detected in tissues of originally defective but spontaneously repaired embryos (results not shown), simultaneously with a completely restored, normal pattern of staining for cadherins and catenins (see Fig. 2G). This suggests that phosphorylation is not likely to be the cause of cadherin and α-, ß-, γ-catenins disappearance. The strong developmental defectiveness was always accompanied with a high dosage of c-Src; overrunning a certain threshold in c-Src expression appeared to be crucial for the development of the defects (Takáč et al., 1998). Thus, it appears that, if Src is in the defective frog embryos involved in the downregulation of the cadherin-catenin system, the downregulation takes place by kinase-independent mechanisms, perhaps via interaction of its domains with not yet identified substrates (compare also Kaplan et al., 1994). Finally, a possibility cannot be excluded that the disappearance of cadherin-catenin complexes reported here might be mediated, by an unknown mechanism, by RSV LTR present in the genomes of the transgenic embryos. As shown here, the process resulting in the defective, low cadherin-catenin phenotype of frog embryos was essentially completed by day 5. Its elucidation and timing will be the subject of our further investigations.

In conclusion, the results presented here provide evidence for the association of aberrant morphogenesis of *X. laevis* embryos expressing a high level of c-Src with the disappearance of cadherin-catenin complexes. The mechanism of the involvement of c-Src overproduction in the loss of the complexes is not clear at present. Further studies should be focused on the assembly of cadherin-catenin complexes, on the regulation of their synthesis and degradation, and on the exchange of their components during embryogenesis.

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