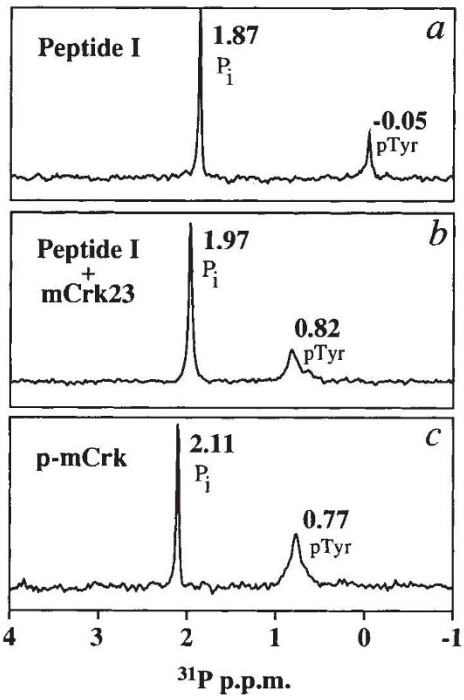


FIG. 3 One-dimensional $^{31}$P spectra of peptide I (*a*), peptide I plus a 50% excess of mCrk23 (*b*), and p-mCrk (*c*) recorded in 50 mM MOPS buffer, pH 6.8, 1 mM sodium phosphate (as an internal pH reference), 200 mM NaCl, 2 mM EDTA, 1 mM DTT, 2 mM benzamidine, 0.02% $NaN_3$. Spectra were recorded at 35 °C at a spectrometer frequency of 202 MHz. Chemical shifts are referenced to external 85% phosphoric acid at 0 p.p.m. The change in chemical shift of the phosphate peak (near 2 p.p.m.) across the three spectra represents a difference of ~0.2 pH units[12].

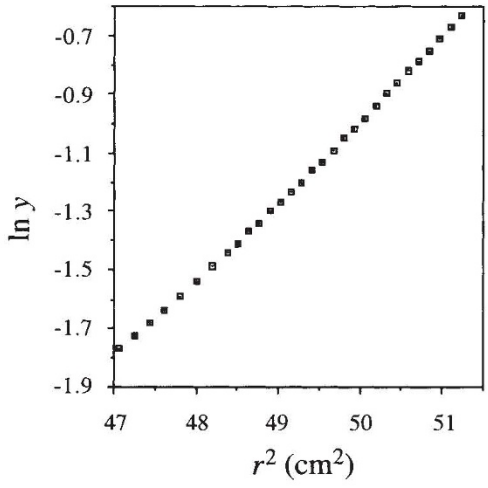


FIG. 4 Phosphorylated Crk is monomeric. A plot of the natural logarithm of the optical density (*y*) as a function of the square of the distance moved in the centrifuge cell ($r^2$). Data were recorded at 37 °C. Similar results were obtained at 20 °C.

METHODS. All hydrodynamic measurements were made in the buffer used in the NMR experiments (Fig. 1 legend). Analytical ultracentrifugation and data analysis are described in ref. 26. Initial sample concentration was determined from a fringe count assuming an average refractive increment of 4.1 fringes mg$^{-1}$ ml$^{-1}$ according to ref. 27. Determination of molecular mass by light-scattering at 20 °C was essentially as described[26]. A series of protein solutions at 1.06–2.73 mg ml$^{-1}$ were injected directly into a Dawn F multiangle laser light-scattering photometer (Wyatt Technology) using a manual injector. The common intercept on a Zimm plot of the extrapolations to zero angle and zero concentration yielded the reciprocal molecular mass; a value of 0.185 was assumed for d*n*/d*c* for the sample.

however, appear to be able to bind these species only in the non-phosphorylated state[5,25]. Thus, the intramolecular SH2–pTyr interaction may also inhibit intermolecular interactions involving the intervening N-terminal SH3 domain. Phosphorylation then provides a mechanism for regulating the SH2/SH3 adaptor function of Crk. The direct identification of an intramolecular SH2–pTyr interaction in p-mCrk also supports the idea that this is a general mechanism by which the activities of SH2-containing proteins, including Src-family kinases, may be regulated. □

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# CORRECTION

## Positional cloning of the mouse *obese* gene and its human homologue

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DURING the course of experiments to define the structure of the mouse *obese* gene, it has come to our attention that the first 58 base pairs of the published 5′ untranslated sequence are not found in the genomic DNA corresponding to the first exon of the gene. It is assumed that the presence of this sequence in one of the complementary DNA clones was the result of a cloning artefact. These nucleotides are in part identical to a sequence in the 3′ untranslated sequence. The ambiguities in the restriction map that resulted from an apparent direct repeat in the 5′ and 3′ untranslated region led us to conclude that the polymorphic *Bgl*II site in SM/Ckc+$^{Dac}$*ob*$^{2J}$ mice was upsteam of the RNA start site. Current data indicate that this polymorphism is the result of genomic alteration in an intron of the *ob* gene (manuscript in preparation). These new data do not affect the coding sequence of the mouse *ob* gene or any of the conclusions in the paper. □