The protein tyrosine kinase, focal adhesion kinase (FAK), is a central regulator of integrin-mediated signaling (Mitra et al., 2005; Parsons, 2003; Schlaepfer and Mitra, 2004). FAK is present in focal adhesions, adhesive structures that mediate attachment of cells to the extracellular matrix (ECM) (Hynes, 1992; Lauffenburger and Horwitz, 1996). In addition, FAK is present in other adhesion structures within the cell, most notably the dynamic adhesion complexes found at the periphery of lamellipodia of migrating cells (Webb et al., 2004). FAK is essential for the turnover (e.g. breakdown) of this latter class of cell adhesions and has also been implicated in the release of adhesion at the rear of the cell (Carragher et al., 2001; Webb et al., 2004). Inhibition of FAK activity or targeted deletion of FAK expression results in cell migration defects (Ilic et al., 1995; Richardson et al., 1997a; Schlaepfer and Mitra, 2004). Increased FAK expression is a hallmark of many cancers and may contribute to the metastatic phenotype of highly malignant cells (Gabarra-Niecko et al., 2003).

Clustering of cell surface integrins, as a consequence of cell attachment to ECM proteins or treatment with certain integrin-specific antibodies, leads to the activation of FAK catalytic activity and an increase in FAK tyrosine phosphorylation (Mitra et al., 2005; Parsons, 2003). Activation of FAK results in autophosphorylation of Y397, the recruitment of Src and Src-family kinases, and the increased phosphorylation of other proteins present in the adhesion complexes, notably paxillin and p130Cas (Mitra et al., 2005). The present evidence suggests that the initiation of this tyrosine phosphorylation cascade is essential for the downstream signaling mediated by cellular adhesion complexes.

In addition to being a protein tyrosine kinase, FAK appears to perform a scaffolding function, binding to a variety of membrane-associated proteins including growth factor receptors and ERM proteins. In adhesions, FAK co-localizes with and binds to the focal adhesion proteins paxillin, p130Cas and talin, as well as SH3-domain-containing GTPase-activating proteins for the Rho and Arf family of small GTPases. In addition, the phosphorylation of specific tyrosine residues appears to be important for the recruitment of SH2-domain-containing signaling proteins, including Src and Src family kinases, PI 3-kinase and Gbr2 (Mitra et al., 2005; Parsons, 2003; Parsons et al., 2000).

Little is known about the spatial and temporal organization of FAK in newly formed or more stable cellular adhesions. Further, little information is available about the factors that regulate the enzymatic activation of FAK or the interaction of FAK with its binding proteins. To understand the possible role of post-translational modifications on the regulation of FAK function, we have mapped the phosphorylation sites within FAK using immunoaffinity purification of FAK and mass spectrometry (MS). In this report, we identify 25 sites of phosphorylation (15 serine, 5 threonine and 5 tyrosine residues), including a number of the serine and tyrosine phosphorylation sites previously reported. In addition, we note the juxtaposition of phosphoserine-, phosphothreonine- and phosphotyrosine-containing residues, which suggests that coordinated phosphorylation of FAK by serine/threonine and tyrosine-specific kinases may be an important aspect of regulation of FAK function.

To identify the sites of phosphorylation in FAK, MS analysis was performed on FAK immune complexes prepared from HEK293 cells expressing chicken FAK. In some cases the cells were pretreated with phosphatase inhibitors to enhance the recovery of phosphorylated residues. Through this analysis, peptides representing 90% of the FAK protein were identified. Fig. 1 shows the sequence of FAK with all of the observed phosphorylation sites marked in red. The amino acid numbering in this report is based on the chicken sequence, which differs slightly from the numbering for human or mouse FAK. The regions of the protein that were not identified by this MS analysis are shown in lower case. The phosphorylation of residues within these regions is therefore unknown. In addition, there were two instances in which the specific phosphorylated amino acids within a peptide could not be assigned by the MS/MS spectra (shown in brackets). A total of 38 phosphopeptides were identified, and their sequences were confirmed by manual inspection of the spectra (Table 1). From these peptides, we have identified the phosphorylation of 15 serine, 5 threonine and 5 tyrosine residues.

Fig. 2 shows the superposition of the detected phosphorylation sites on the domain structure of FAK. A number of features of the phospho-landscape are notable. The N-terminal FERM domain serves an autoinhibitory function (Cooper et al., 2003; Dunty et al., 2004) and as a bridge to growth factor receptors and membrane adapter proteins such as ezrin (Poulet et al., 2001; Sieg et al., 2000). Two sites of phosphorylation, T13 and S29, were observed N-terminal to the beginning of the FERM domain. Both residues are conserved in FAK from man, mouse and frog, indicating conservation of function in this region (Table 2). This region is unique to FAK and does not share sequence similarity with the related FAK kinase, PYK2/CAKβ. In the middle of the FERM domain, Y155 is a site of phosphorylation. Y155, which is conserved in FAK from man, mouse and frog, as well as in PYK2, is proximal to the previously identified site of FAK sumoylation, and phosphorylation of this residue may play a role in regulating nuclear trafficking of FAK (Kadare et al., 2003).
identified sites of phosphorylation (S386, T388, S390, S392 and T406), as well as Y407, which was previously identified as a site of Src phosphorylation (Calalb et al., 1995). S390, S392 and T406 are conserved amongst FAK proteins from mouse, man and frog, whereas S386 and T388 are chicken specific. The proximity of this cluster of phosphorylation sites to Y397 suggests that phosphorylation of these residues plays a role in the regulation of FAK enzymatic activity. Current evidence indicates that FAK autophosphorylation takes place via the autophosphorylation of this cluster of phosphorylation sites to Y397. Phosphorylation of Y397 is thought to play a role in the regulation of FAK enzymatic activity (Buck et al., 1998). Y397 constitutes a docking site for the SH2 domain of Src and Src family kinases (Schaller et al., 1994). It is possible that phosphorylation of Y397 may influence the association of FAK with Src or other binding partners that use phospho-Y397 as a docking site.

One new site of phosphorylation was identified in the kinase domain. Y570 is conserved in FAK from man, mouse and frog, as well as in PYK2. It is located proximal to Y576 and Y577, both of which reside within the catalytic lip of FAK and have been reported to be important for the adhesion-dependent activation of FAK (Calalb et al., 1995).

Two additional clusters of phosphorylation sites were isolated within the C-terminal region of FAK, where 14 sites of phosphorylation were mapped (11 serines, 2 threonines and 1 tyrosine). Two of these newly identified sites, T700 and S708, surround a potential site of caspase cleavage (Gervais et al., 1998) and reside just downstream of the acidic amino acid linker region that separates the kinase domain from the C-terminal domain. Both sites are conserved in human, mouse and frog. In addition to these two residues, phosphorylation of S722, S725/726 and S732 were mapped proximal to site I, the proline-rich site of the proximal serine/threonine phosphorylation may be influenced by the proximal serine/threonine phosphorylations. In addition, phospho-Y397 may influence the association of FAK with Src or other binding partners that use phospho-Y397 as a docking site.

Isolated phosphorylation sites were observed at S766, which is conserved between species and lies adjacent to another caspase cleavage site (Gervais et al., 1998), T793 (not conserved) and S845, which is phosphorylated by PKA (Richardson et al., 1997b).

![Fig. 1. Identified sites of serine, threonine and tyrosine phosphorylation in FAK. The sequence of chicken FAK (GenBank accession no. M86656) is shown with the amino acids that were identified by mass spectrometry (MS) shown in capital letters. The phosphorylated residues identified by the MS analysis are shown in red. There were two peptides in which the phosphorylated residue could not be determined (indicated by brackets). Because T394 was only observed within a peptide containing Y397, a known site of phosphorylation, the phosphorylation status of T394 is questionable.](image)

![Fig. 2. Distribution of phosphorylation sites in FAK. The domain structure of FAK is shown with the mapped sites of serine (blue stars), threonine (green stars) and tyrosine (red stars) phosphorylation shown below. The sites of sumoylation (•) and caspase cleavage (*) are indicated.](image)
Finally, a cluster of phosphorylation sites were observed proximal to site II and the beginning of the focal adhesion targeting (FAT) domain. Site II is the proline-rich domain that interacts with SH3 domains from ASAP1 and Graf (Hildebrand et al., 1996; Liu et al., 2002), whereas the FAT domain interacts with a number of focal adhesion proteins including paxillin, talin and Grb2 (Chen et al., 1995; Hildebrand et al., 1995; Schlaepfer et al., 1994). This cluster of phosphorylated residues includes S888, S891, S894, S898, Y899 and S911. S888 is present only in chicken and human FAK; S891 and S898 are conserved in human and mouse; and S894, Y899 and S911 are conserved from human to frog. Ser911 (S910 in human and mouse) is the only site from this group that has been characterized and is reported to be phosphorylated in an ERK-dependent manner (Hunger-Glaser et al., 2003). Because of the large number of phosphorylation sites in this region, it is likely that differential phosphorylation by several different kinases modulate the binding of proteins to this region.

Several features of the MS analysis allow an approximation of the abundance of the different phosphorylated peptides. Within an individual experiment, the relative phosphopeptide abundance can be expressed in terms of ion counts (peak heights) observed for the most abundant charge state. Assigning ion counts for the most abundant phosphopeptides as ++++, peptides that exhibited ion currents decreased by a factor of 10, 100 and 1000 can be indicated by ++, +, and +, respectively (Table 1). Using this method of pseudo-quantitation, the abundance of peptides identified in FAK prepared from cells treated with or without phosphatase inhibitors was

| Residue | FAK peptide sequence (chicken) | (+) Inhibitors | (-) Inhibitors |
|---------|--------------------------------|----------------|----------------|
| T13     | DPNLNHTPSSSAK                   | ++             |                |
| S29     | THLGTGEMSPGAMERVLK              | +++           | ++++           |
| Y155    | NDYMELADQVDQEIALK               | +++           |                |
| S386/T388 | QGVRSHTVSSETDDYAEIIEDE       | IMAC          |                |
| Y397    | QGVRSHTVSSETDYYAEIIEDE         | IMAC          |                |
| S390    | QGVRSHTVSSET                      | IMAC          |                |
| S390[T394-Y397] | QGVRSHTVSST [TDDY] AEIIEDE | IMAC          |                |
| [T394-Y397] | QGVRSHTVSST [TDDY] AEIIEDE | IMAC          |                |
| Y397    | QGVRSHTVSSETDDYAEIIEDE         | IMAC          |                |
| T406/Y407 | DTTYMPSTRDY                  | IMAC          |                |
| Y570    | DFGLSRYME                       | IMAC          | +              |
| T700/S708 | RMRAMESRQQRTVSWDGSQSD        | IMAC          |                |
| S722[S725-S726] | DEPPPKPSRPYPSPR (SS) EGF | IMAC          |                |
| S726    | SHGIPAMAGSIYPGQASLL            | IMAC          |                |
| T793    | DSGTLVDRGMQQLPHTLM             | IMAC          |                |
| S845    | RFLVMPDQVRPLSREGISE           | IMAC          |                |
| S888    | KPPRPGAPHLGLASLNPSPV           | IMAC          |                |
| S888/S891 | KPPRPGAPHLGLASLNPSPV          | IMAC          |                |
| S888/S891/S894 | KPPRPGAPHLGLASLNPSPV   | IMAC          |                |
| S888/S894 | KPPRPGAPHLGLASLNPSPV          | IMAC          |                |
| S888/S894 | KPPRPGAPHLGLASLNPSPV          | IMAC          |                |
| S894    | KPPRPGAPHLGLASLNPSDVSNYEVK     | IMAC          |                |
| S894/S898 | KPPRPGAPHLGLASLNPSDVSNYEVK   | IMAC          |                |
| S894/S898/Y899 | KPPRPGAPHLGLASLNPSDVSNYEVK | IMAC          |                |
| S894/Y899 | LNSPVDVSYEVK                  | IMAC          |                |
| S911    | IKQIEISPFTANL                  | +++           | ++++           |
| S911    | IKQIEISPFTANLDRSNDK            | +++           |                |

Each of the phosphopeptides identified from FAK is listed, with the phosphorylated residue(s) shown in red. The phosphorylated amino acids in brackets could not be distinguished. In some experiments, the cells were treated with phosphatase inhibitors before lysis. All of the cells were lysed in the presence of inhibitors. Relative phosphopeptide abundance is expressed in terms of ion counts (peak heights) observed for the most abundant charge state. Ion counts for the most abundant phosphopeptides are displayed as ++++, those that exhibit ion currents decreased by a factor of 10, 100 and 1000 are shown as ++, +, and +, respectively. The relative abundance of peptides can only be compared within an individual experiment and, thus, peptides that were identified under different conditions may have been present at different levels in different experiments. We have indicated the highest relative amount that was observed. Peptide abundance with ‘+’ inhibitors cannot be compared to abundance without ‘−’ inhibitors. In some cases, peptides were only observed after enrichment with immobilized metal-affinity chromatography (IMAC), indicating that these peptides were present in limited amounts.

Table 1. Relative abundance of phosphopeptides identified by mass spectrometry
The sequence of chicken FAK was aligned with the sequences from human (GenBank accession no. M95408) and the frog, *Xenopus laevis* (GenBank accession no. L33920) by the Clustal method using the DNASTAR MegAlign program. The phosphorylated amino acids that are identical are shown as ‘+’. The numbering of residues was based on the sequence of chicken FAK and may vary slightly for other species.

| Residue | Human | Mouse | *Xenopus* |
|---------|-------|-------|-----------|
| T15     | +     | +     | –         |
| S29     | +     | +     | +         |
| Y155    | +     | +     | +         |
| S386    | –     | –     | –         |
| T388    | –     | –     | –         |
| S390    | +     | +     | +         |
| S392    | +     | +     | +         |
| [T394]  | +     | +     | +         |
| Y397    | +     | +     | +         |
| T406    | +     | +     | +         |
| Y407    | +     | +     | +         |
| Y570    | +     | +     | +         |
| T700    | +     | +     | +         |
| S708    | +     | +     | +         |
| S722    | +     | +     | +         |
| [S725]  | +     | +     | +         |
| [S736]  | +     | +     | +         |
| S732    | +     | +     | +         |
| S766    | +     | +     | +         |
| T793    | –     | –     | –         |
| S845    | +     | +     | +         |
| S888    | +     | +     | –         |
| S891    | +     | +     | +         |
| S894    | +     | +     | +         |
| S898    | +     | +     | +         |
| Y899    | +     | +     | +         |
| S911    | +     | +     | +         |

The sequence of chicken FAK was based on the sequence of chicken FAK and may vary slightly for other species.

### Materials and Methods

HEK293 cells expressing low levels of FLAG-epitope tagged FAK (one- to twofold greater than endogenous levels) were grown on 135 mm cell culture dishes (Nunc). The cells were treated for 30 minutes with a serine/threonine phosphatase inhibitor calyculin (0.1 μM) and/or a tyrosine phosphatase inhibitor sodium orthovanadate (100 μM), or they were processed without any inhibitor treatment. FLAG-FAK was captured from clarified cell extracts made in CSK-NP buffer [150 mM NaCl, 5 mM MgCl₂, 20 mM Tris-HCl (pH 7.4), 1 μM orthovanadate, 2 mM sodium pyrophosphate, 1% NP-40, 1% EDTA-free protease inhibitors (Roche)] using M2-agarose affinity resin (anti-FLAG antibody conjugated to agarose, Sigma). The M2 agarose beads were sequentially washed with CSK-buffer, MS buffer (150 mM NaCl, 5 mM MgCl₂, 20 mM Tris-HCl pH 7.4) and 1 M buffer (1 M NaCl, 5 mM MgCl₂, 20 mM Tris-HCl, pH 7.4) in order to remove non-specific binding proteins. The FAK protein was eluted with 0.3 mg/ml FLAG peptide in MS buffer. Typically, 30–40 pmol of purified FAK was recovered from 10⁶ cells, and 2-10 pmol of protein was used to initiate C18 reverse phase (RP)-HPLC analysis and mass spectrometry. Aliquots of purified FAK protein were digested with either GluC, LysC or AspN or a combination of LysC and AspN to maximize the coverage of peptides analyzed in the mass spectrometer. The resulting peptides were separated by RP-chromatography coupled to online detection by nanoflow LC and microcapillary ESI tandem mass spectrometry (LTQ-FTMS, Thermo Electron, San Jose, CA). In some cases enrichment of phosphopeptides was performed with immobilized metal affinity chromatography (IMAC) (Ficarro et al., 2002), prior to RP-chromatography. In those cases, 10-30 pmol of protein were loaded onto IMAC columns.

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