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Hormones, cytoskeletal proteins and cell shape

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Recently attention has been focussed on the molecular mechanisms involved in cell shape changes brought about by chemical signals through cell surface proteins. The shape of any given cell type is maintained by various components of the cytoskeleton (microfilaments, intermediate filaments and microtubules), the extracellular matrix, the plasma membrane and associated proteins. Hormones and growth factors regulate the expression and post-translational modifications of these proteins which in turn depend on the phenotype of the cell. Action of hormones and growth factors on cells is also dependent on various factors such as phase and age of the cell, ion gradient and presence or absence of specific receptors at the right sites. The cause and effect in regulation of cell shape is not yet very clear.

THE change in cell shape can be considered as a primary adaptation of the cell to suit altered physiological demands. Cell shape changes are manifestations in many of the cellular events such as division, differentiation, transformation and death. A variety of observations suggest that cell shape changes exert specific effects on gene expression[1]. The exact mechanisms by which changes in cell shape affect the pattern of gene expression are not yet very clear but the cytoskeleton seems to play a key role in this process[2–4]. To understand functional and spatial relationship of the cell in a given tissue, it is necessary to know how the local microenvironment acts on the cell to regulate its phenotype. Cell phenotype is controlled by restructuring the cytoskeletal framework which in turn depends on interactions with neighbouring cells and extracellular matrix (ECM)[5–8]. A host of extracellular and intracellular proteins are involved in shaping the cellular architecture. The extracellular matrix proteins such as collagen, fibronectin, laminin, proteoglycans and fibrinogens[9], the transmembrane proteins such as integrins, cadherins[10], the cyto-

plasmic cytoskeletal proteins, microfilaments (MFs), intermediate filaments (IFs) and in some instances microtubules (MTs) which are sites for immobilizing signaling molecules, regulatory enzymes and metabolic substrates transduce the message to the nucleus[11]. Various factors namely hormones, growth factors (GFs), tumour promoters are known to regulate the expression and/or the activity of many of the above mentioned proteins. These hormonally regulated proteins modulate cytoarchitecture of the target cells[12–15]. Altering the cytoskeletal structure may in turn change the availability of regulatory and catalytic sites of key signal transducing molecules.

During the various physiological processes, the cells undergoing altered cell morphology receive signals from the exogenous chemical factors and through different cell-signalling molecules present at the membrane and in the cytoplasm the information is transmitted to the nucleus where in coordination with internal signals, induction of specific gene expression takes place[16,17].

The study of mechanisms involved in the regulation of cell shape change and gene expression by various factors is an active field of research. Hormones and GFs are known to affect the expression, synthesis and post-translational modifications of cytoskeletal proteins. Plasma membrane fluidity is also known to change due to hormone treatment and change in fluidity is known to affect surface projections and regions of attachment of cytoskeletal elements to plasma membrane[18,19]. Modulation of cytoskeletal proteins and plasma membrane constituents by hormones is responsible for change in cell shape followed by altered gene expression. In attempting to understand the genomic effect brought about by cytoskeletal proteins, Puck and Krystosek[20] have shown that during reverse transformation there is an increased sensitivity of DNA to hydrolysis by DNase I in the presence of the factors such as cyclic AMP, retinoic acid and nerve growth factor (NGF). They observed in the ovary derived normal fibroblasts and cyclic adenosine monophosphate (cAMP) reverse transformed chinese hamster ovary cells (CHO-K1) a clear region of DNase I sensitive DNA around the nuclear periphery. In malignant cells specific differentiation genes were sequestered and therefore could not function and exposed regions were not observed. It was also found that the cytoskeletal disorganizing agents such as colcemid and cytochalasin B prevented the basic feature of reverse transformation in enhanced genome exposure, that is closely linked to the transcriptional activation of certain genes. Cytoskeletal proteins such as the IFs and nuclear lamins seem to be actively involved in increased genome exposure, bringing about altered cell morphology.

The various factors (such as hormones) affecting cytoskeletal organization and the non-genomic and genomic pathways involved in bringing about alterations in cell shape are briefly described in this review.

Regulators of cell shape

An analysis of mechanism of shape determination reveals that the ECM and cytoskeleton are the principal determinants of cell shape[5,7,8,21]. The ECM by itself can influence the shape of a cell. Cells grown on ECM substrate have cell shapes, proliferation rates and responses to GFs which differ from cells grown on plastic or glass[22]. For instance, granulosa cells cultured on plastic become flattened in shape and develop stress fibres but when grown on ECM the cells become round and closely resemble their counterparts *in vivo*[12]. Similarly mouse mammary epithelial cells become cuboidal and increase milk protein synthesis when grown on floating collagen gels[23] as compared to cells on plastic or collagen coated plastic[24]. It is now generally accepted that there is a physical link between the extracellular matrix and the cytoskeleton. The link is established with the interactions of the ECM via their cell surface receptors to the actin cytoskeleton. Many of the receptors for ECM proteins such as collagen[25,26], fibronectin and their receptors integrins[10] and laminin[27] have been isolated and these receptors are linked to the actin cytoskeleton within the cell. Fibronectin and collagen are known to be involved in hormone-induced responses[28,29].

Changes in cell shape also involve reorganization of the cytoskeletal elements – MFs, MTS and IFs. The use of inhibitors of cytoskeletal protein polymerization such as cytochalasins, colcemid, vinblastine and nocodazole has proved beyond doubt that MFs and MTs are actively involved in change in cell morphology[30–33]. Many hormones, growth factors and tumour promoters are known to alter cell morphology by affecting the assembly/disassembly of cytoskeletal proteins[12,14,32,34].

The ability of actin to polymerize/depolymerize enables the cell to rearrange the MF organization. Anchorage-dependent cells adhere tightly to the underlying substratum through focal adhesions. In many cultured cells, large bundles of MFs are prominent at focal adhesion points[35]. Many characteristics reveal that they are structurally and functionally equivalent to adhesions made by the cell to ECM *in vivo*[36]. The actin cytoskeleton is linked to plasma membrane proteins, adhesion plaque proteins and to cytoskeletal proteins (Figure 1). Factors affecting actin or actin-associated proteins can also bring about change in organization of the cell.

The rounding of granulosa cells on treatment with follicle stimulating hormone (FSH) was reported to be associated with down-regulation of synthesis of adherens junction proteins, namely α-actinin, actin and vinculin[12]. Lomri and Marie[14,34] found that parathyroid hormone (PTH) elicited an increase in synthesis of actin in cultured mouse osteoblastic cells. Actin assembly/disassembly was found to be regulated by the expression of actin and vinculin by a feedback loop in 3T3 and HeLa cell lines. In cells which showed elevated

levels of depolymerized actin, the actin mRNA was found to be reduced[37].

The IFs are also known to be involved in cell shape changes. For example, upon stimulation of MCF-7 cells (human breast cancer cell line) by estradiol an increase in the keratin filament network results into cytoplasmic ridges on the cell surface[38,39]. In primary cultures of rat vaginal epithelial cells (VEC) on addition of estradiol, Vijaysaradhi *et al.*[40] found long microridges on the cell surface which are characteristic of cornified cells indicating increased keratin network just below the plasma membrane. Phorbol esters known to mimic hormone action also bring about cell shape changes during transformation. A reorganization of cytokeratins along with disruption of junctional complexes was observed when Madin-Darby bovine kidney (MDBK) cells were treated with tetradecanoyl phorbolacetate (TPA)[41]. Microtubular rearrangement was found in rat phaeochromocytoma cells upon activation by NGF[42]. Tubulin synthesis increased by two-fold in response to NGF whereas the synthesis of microtubule associated protein (MAP) and Tau increased 20-fold. Increased synthesis of tubulin and other MAP proteins are associated with changes in organization of MTs[43]. These studies suggest a link between changes in cell growth, differentiation, configuration and cytoskeletal protein synthesis.

Regulation of cytoskeleton and associated proteins

Extracellular and membrane proteins

The cytoskeletal elements are in close association and are capable of interacting with each other and with the transmembrane proteins. The ECM transduces a series of signals to the nucleus through cytoskeletal elements. This mechanism of signal transduction may be the result of mechanochemical and/or biochemical processes. In the mechanochemical process the cytoskeleton may regulate gene expression by interacting with the nuclear matrix which may lead to physical expansion of nuclear pores, thereby increasing the rate of nuclear transport in

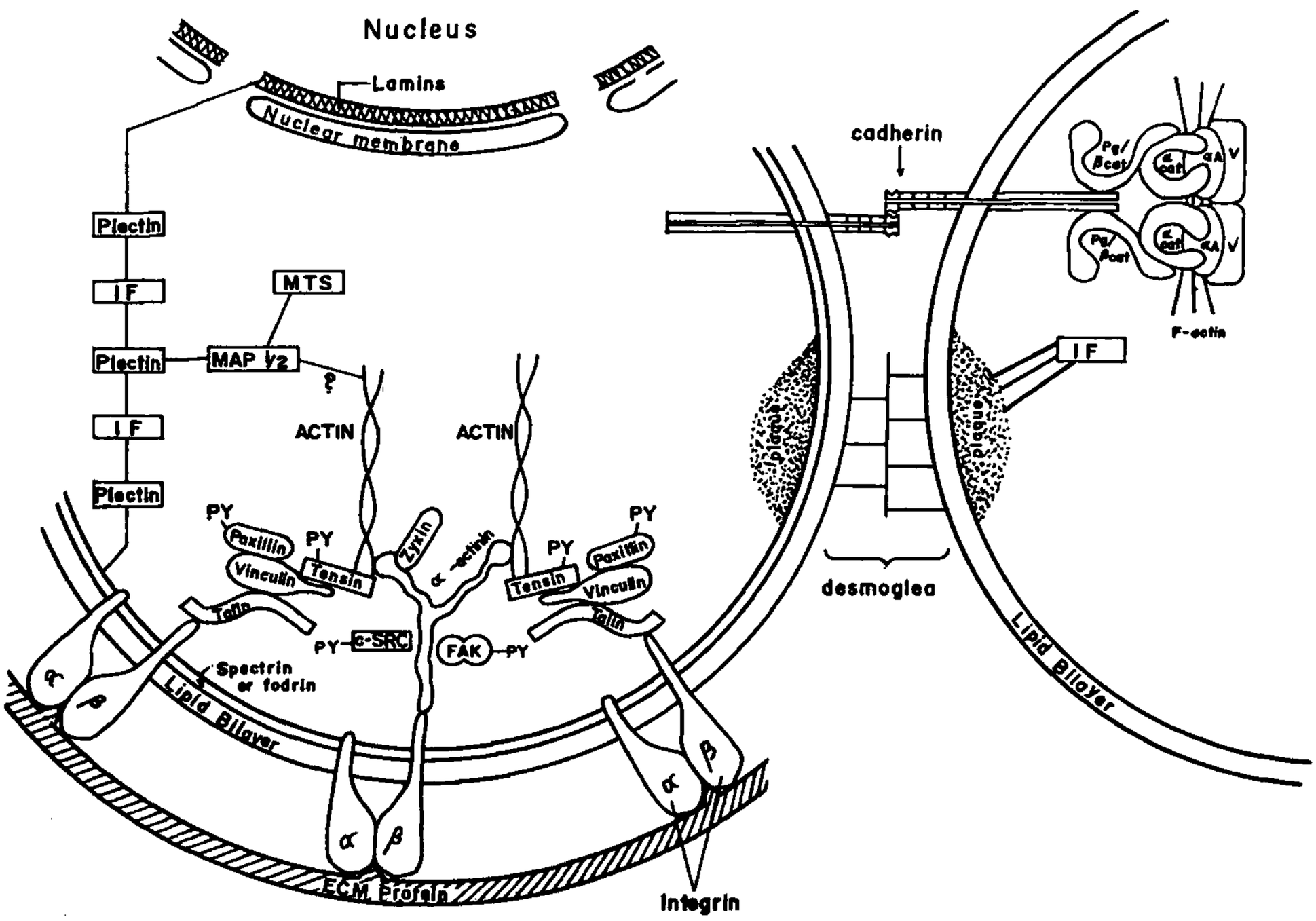

Figure 1. Schematic diagram of two cells depicting the association between cytoskeleton and ECM at focal adhesions; the desmosomes which form another link between two adjacent cells are connected to the cytokeratin IF. Integrins, the receptors for ECM proteins such as fibronectin are connected to actin filaments through two distinct linkages. Talin–vinculin–tensin form one bridge while α-actinin forms another. The actin filaments appear to be connected to IFs and MTs via the MAPs and IFAPs (plectin). The cadherins which connect two cells are linked to actin via the β-catenin/plakoglobin-α-catenin and α-actinin. FAK, focal adhesion kinase; PY, phosphotyrosine; IF, intermediate filament; MT, microtubule; MAP, microtubule associated proteins; Pg, Plakoglobin; βcat, β-catenin; αcat, α-catenin; αA, α-actinin; V, vinculin.

spreading cells[44]. Signals from the ECM reach the nucleus by a series of enzymatic events at the plasma membrane level which in turn involve cytoskeletal elements to convey the message to the nucleus. Among the prominent plasma membrane proteins associated with actin are the cadherins and integrins. Cadherins, calcium dependent-cell adhesion molecules, are transmembrane proteins that interact with cytoplasmic proteins called catenins (α and β-catenins). Catenins link cadherins to the actin cytoskeleton[45] (for more details, see the legend of Figure 1). It is evidenced by the fact that the cells which express cadherins but lack catenins are of non-adherent type[46,47]. Cadherins through interactions with catenins form adherens type cell junctions in epithelial cells[48,49]. β-catenin or plakoglobin constitutes a bridge between cadherins and N-terminal domain of α-catenin. α-catenin then connects this membrane-associated complex with actin cytoskeleton either directly or indirectly via α-actinin[50]. It is suggested that the control of equilibrium levels between free and bound pools of catenins could play a role in regulating cellular responses to extra-cellular signals for cell–cell adhesion or cell proliferation. It seems the phosphorylation and dephosphorylation of catenins regulate the ratio of free and actin bound cadherin–catenin complexes, thereby regulating the adhesive forces of cadherins[51]. Desmosomal cadherins, the desmocollin and desmoglein are connected to the IFs via desmplakin and band 6 protein in contrast to other cadherins which are connected to actin filaments directly[50].

Integrins which act as receptors for many extracellular proteins, such as collagens, laminin, entactin, fibronectin, vitronectin and fibrinogen, are associated with the actin cytoskeleton inside the cell[52]. Integrins appear to be connected to actin filaments through two distinct linkages. Talin, vinculin and the actin-capping protein tensin form one bridge, while the actin filament cross-linking protein α-actinin forms another (Figure 1; see refs 52, 53). The integrins are essential for cell adhesion both *in vitro* and *in vivo*[54,55]. Integrins have a role as signalling receptors where they effect the release of second messengers due to hydrolysis of phosphotidylinositol 4,5 biphosphate (PIP_2) which requires both adhesion of cells to ECM and binding of platelet derived growth factor (PDGF) to its receptors. Clustering of integrins in focal adhesions triggers activation of a phosphotidylinositol phosphate (PIP)-5-kinase that catalyses phosphorylation of PIP to PIP_2 and clustering of PDGF receptors by PDGF triggers the activation of phospholipase C (PLC). When cells receive both these stimuli, significant breakdown of PIP_2 occurs, causing release of second messengers such as inositol triphosphate (IP_3) and diacylglycerol (DAG) (Figure 2)[52,56]. Streuli *et al.*[57] demonstrated that mouse mammary epithelial cells can direct β-casein gene expression in the presence of prolactin (PRL) and laminin, a component of the basement membrane. A cooperative signalling through integrins and PRL receptor is necessary for the differentiated phenotype. These examples demonstrate that adhesion to ECM proteins enhances responsiveness of certain cells to hormones.

Adhesion plaque proteins

The expression and post-translational modifications of adhesion plaque proteins such as vinculin and α-actinin regulate cell adhesiveness. Decreased expression of vinculin or its phosphorylation may result in an inefficient assembly of adhesion plaque proteins leading to decreased adhesion[12,58]. On the other hand, increased synthesis of vinculin may facilitate more efficient recruitment and assembly of adherens junction proteins leading to increased tightness of adhesion and decreased motility of the cell[59]. α-actinin known to cross-link MFs is also involved in binding actin filaments to the membrane. Increased level of α-actinin at the junctions leads to more stable MF–membrane interaction and hence increased tightness of adhesion[60].

Many extracellular factors induce phosphorylation of focal adhesion proteins at tyrosine residues[61,62]. For example, binding of cells to fibronectin and stimulation by neuropeptides results in tyrosine kinase $p125^{FAK}$ (focal adhesion kinase) phosphorylation[63]. Substrates for activated $p125^{FAK}$ include paxillin and tensin, the components of focal adhesion plaques. Overexpression of FAK-C-terminal domain suggests that FAK may regulate focal adhesion assembly via protein–protein interactions[64,65].

Microfilament proteins

Cell surface appendages like microvilli and microridges are supported mainly by MF proteins such as actin. Actin MFs are prominent at focal adhesions and are linked to integrins in the plasma membrane via talin, vinculin, tensin, α-actinin and zyxin[52,66]. Structural changes in actin and plasma membrane components during cell division, transformation and cell death bring changes in cell shape. Luciano *et al.*[67] have shown that during the apoptotic programme of enterocytes, the MFs supporting the microvilli at the terminal web region and zonula adherens are withdrawn due to depolymerization of actin and therefore bands of microvilli are detached from main cell body and cell attains a rounded morphology.

During the G-1 phase of the cell cycle, certain types of cells in culture show large number of microvilli, blebs and ruffles which diminish on the onset of S phase and finally the cells become relatively smooth. The microvilli increase in number during G2 phase as cells thicken in anticipation of rounding up for mitosis. MFs reorganize as cells round up for mitosis and provide

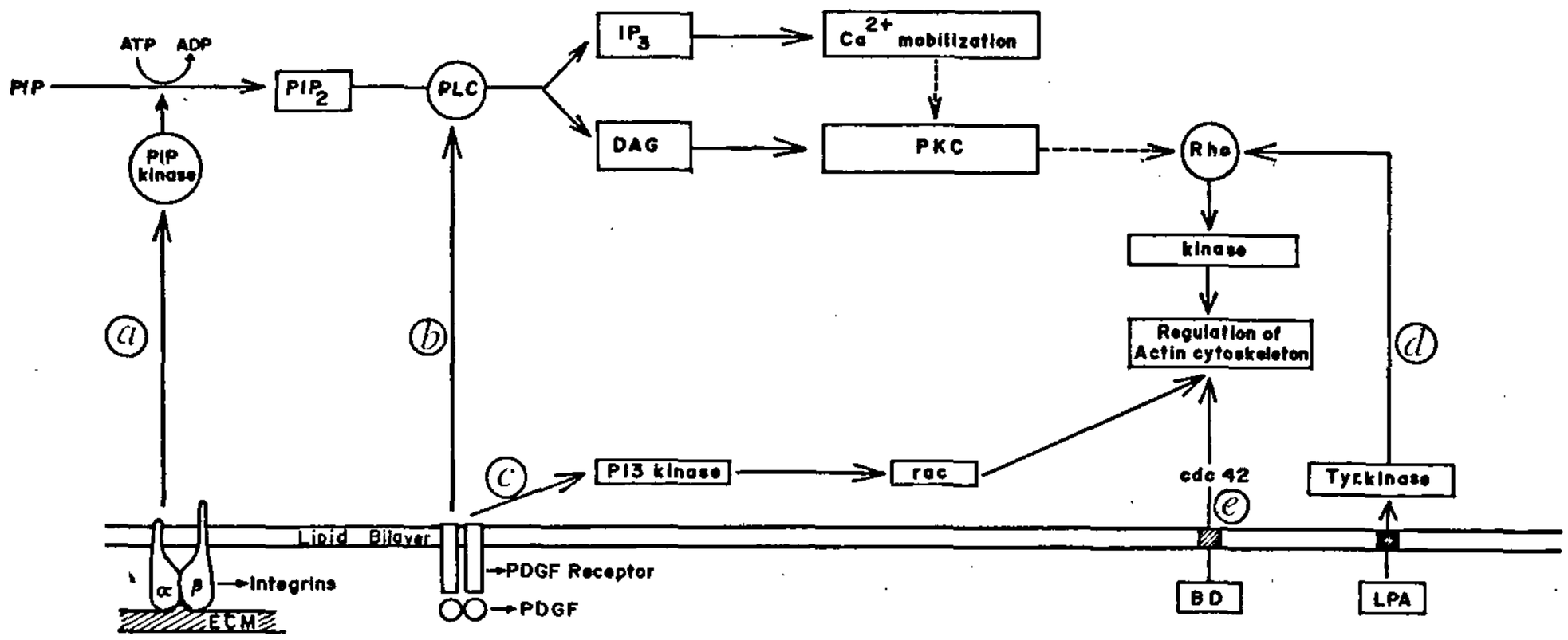

Figure 2. The various signal transduction pathways regulating actin cytoskeleton in Swiss 3T3 fibroblast cells. The ligand–receptor complex transduces the message to the rho family of small GTP-binding proteins via specific kinases. Some of these kinases are phosphotidylinositol 3-kinase (PI3K), protein kinase C (PKC) and tyrosine kinase. *a*, Adhesion of cells to ECM via the clustering of integrins triggers PIP kinase resulting in increased levels of PIP_2; *b*, Binding of PGDF to its receptor activates PLC resulting in calcium mobilization and PKC activation. Rho may be present downstream of PKC regulating actin polymerization; *c*, PGDF and insulin stimulate rac via PI3K which affects actin polymerization at the region of membrane ruffles; *d*, Lysophosphatidic acid (LPA) regulates rho via tyrosine kinase which affects actin polymerization at the region of stress fibres; *e*, Bradykinin (BD) acting via cdc42 affects actin in filopodia[79]. Upstream of each rho family member guanine nucleotide exchange factors are needed which keep them in active form and the GTPase activating proteins (GAP) negatively regulate their activity.

contractile force during cytokinesis[68]. So also mitogenic hormones such as estradiol prepare the cells to divide by causing the cells to shed off microvilli and cells round off in the basal layer of vaginal epithelium of rat[4,69]. During the process of differentiation, cuboidal basal cells transform to flattened cells with an increase in fluidity of the plasma membrane[70]. During both the processes, cell division and cell death, cell surface appendages are withdrawn, the contact points between plasma membrane and actin fibres are reduced and MF network collapses[71]. The levels of cholesterol and proteins on the membranes are reduced and membrane becomes more fluid[72].

In *in vitro* experiments, stimulation of quiescent serum starved Swiss 3T3 fibroblast cells by various GFs, bradykinin or lysophosphatidic acid result in rearrangement of F-actin[73]. Such kind of cellular responses at the plasma membrane seem to constitute distinct signalling pathways controlled by Rho family of small GTP-binding proteins such as rho, rac or cdc42 inducing formation of stress fibres, membrane ruffles or filopodia (Figure 2)[74]. There is an evidence that stimulation of rac by PDGF and insulin is mediated by PI3 kinase[75]. A tyrosine kinase appears to be required downstream of rho since rho-mediated induction of stress fibre formation in Swiss 3T3 cells is inhibited by tyrosine kinase inhibitor genistein[76–79]. Rho is also found to regulate enzymes such as phosphoinositide-3-kinase and phosphotidyl inositol-4-phosphate-5 kinase. Hence rho is involved in regulation of actin cytoskeleton through the formation of phospholipid intermediates[80].

Thus several signals induced by growth factors, hormones and tumour promoters have been implicated in regulating actin cytoskeleton. The classic second messengers IP_3 and DAG produced by hydrolysis of PIP_2 seem to play a role in the signalling cascade. DAG stimulates protein kinase C (PKC) and pharmacological activators of PKC, phorbol esters stimulate actin reorganization in fibroblasts[77,81]. One of the isoforms of PKC[82] and various other kinases of the src family and PLC have also been localized to adherens junctions. Events like PKC activation by hormones or GFs involve reorganization of vinculin, depolymerization of actin and phosphorylation of certain proteins that are important for stabilization of the MF-membrane interaction (Figure 2). Woods *et al.*[83] have found that PKC activation may play an important role in focal adhesion formation in the human embryo fibroblasts. IP_3 stimulates release of calcium from intracellular stores and the activity of several actin binding proteins *in vitro* has been shown to be regulated by calcium concentration[84]. PIP_2 itself binds to a number of actin-binding proteins *in vitro* and inhibits their interaction with actin[85] leading to the proposal that alterations in PIP_2 levels could modulate actin organization *in vitro*[86]. A linkage between actin-binding proteins and PIP_2 pathway has also been sug-

gested by Ben-Ze'ev[1]. Extracellular factors alter adenylate cyclase activity leading to change in the level of cAMP and modulating activity of protein kinase A (PKA). Activators of PKA stimulate the dissolution of stress fibres in fibroblasts, suggesting another signalling pathway in actin organization[87].

Intermediate filament proteins

In the foregoing discussion, the actin cytoskeleton is viewed extensively as a signal transducer, the organization/disorganization of which leads to changes in cell morphology and altered gene expression. It is also well known that the three cytoskeletal elements the MFs, IFs and MTs are interconnected to form a cytoplasmic network. The intermediate filament-associated protein (IFAP), plectin, has been shown to localize to focal adhesions where it seems to function as a cross-linker between IFs and actin[88].

Various experiments demonstrate that the organization of IFs is affected by hormones and growth factors, resulting in altered cell morphology and gene expression. Keratin IFs generally expressed in cells of epithelial origin, represent the most complex group of proteins in IF family. *In vivo* studies done in our laboratory suggest that the aggregation and dispersion of keratin filaments during the differentiation of rat VEC in the presence of estradiol is a consequence of successive cycles of phosphorylation and dephosphorylation of keratin polypeptides[3]. Modulation in intracellular levels of calcium depends on estradiol levels[89,90] and the association of calcium-dependent cross-linking enzyme transglutaminase (TGase) with the IFs[91]. Keratins become relatively rigid due to phosphorylation which may facilitate cross-linking of the assembled keratins by disulphide linkages and Σ(τ-glutamyl) lysine bonds leading to terminal differentiation of keratinocytes which assumes flattened morphology[90,92]. Several other groups[93,94] have demonstrated the involvement of filaggrin in the aggregation of phosphorylated keratins into filament bundles in epidermal cells.

Retinoids and progesterone diminish features of terminal differentiation and convert the cell to the secretory type. Removal of retinoids prepares the cells for terminal differentiation by inducing keratin expression at transcriptional level[95–97]. Many growth factors also act as modulators of IF network. EGF, transforming growth factor (TGF), keratinocyte growth factor (KGF) and cytokines can also induce alterations in cytoarchitecture. NGF is a known inducer of neurofilaments (NFs) and peripherin expression in developing neurons. Phosphorylation of NFs plays an important role in development, cross-linking and stability of the filament network with axons, this in turn regulates interactions of IFs with each other or with other cell components[98,99]. Vimentin also undergoes alternate phosphorylation and dephosphorylation with a looser network resulting from phosphorylation. In MCF-7 cells, a looser network of vimentin type IF is seen on estradiol treatment. It has been shown that norepinephrine induces conversion of flat sertoli cells into stellate morphology resulting from phosphorylation of vimentin[100]. Ben-Ze'ev[41] followed the regulation of synthesis of cytokeratin and desmoplakin, the proteins involved in the construction of desmosomal junctions. Along with the disruption of the desmosomes and reorganization of cytokeratins there was a dramatic decrease in the synthesis of cytokeratins and desmoplakin in the TPA-treated MDBK cells. However, Gupta *et al.*[4] found that during keratinization of VEC, cellular connections were many fold higher in estradiol primed immature rats with increased keratin synthesis in the intermediate layers. The regulation of vimentin type IF which is coexpressed in MDBK cells was different from that of keratins and this may be due to spreading of cell on the substrate.

Microtubule proteins

Rosette and Karin[101] have provided evidence to show that MT depolymerizing agents activate sequence-specific transcription factors such as NF-kappa B (NF-KB) and induce NF-KB dependent gene expression in HeLa cells. In the unstimulated HeLa cells they found that majority of the NF-KB resided in the cytoplasm as a complex with its inhibitor I kappa B (IKB). Upon stimulation with nocodazole the NF-KB translocates to the nucleus with a concomitant degradation of IKB. It is known that NF-KB activity is induced in presence of TPA IL-1 and certain growth factors[102] and all these agents are also known to reorganize the cytoskeleton[103]. Hence there is a possibility that selective depolymerization of MTs by any of these agents could be an intermediate in the signalling pathway, leading to activation of NF-KB in turn modulating gene expression to give a differentiated phenotype.

Possible interaction between cytoskeletal proteins and genes

Recently a few authors have described the probable mechanism of mammalian gene regulation by the cytoskeletal elements[20,104]. Gene regulation in mammalian cells seems to operate at two levels. The first step is the activation of tissue-specific genes in their conversion from the sequestered to the exposed state. This involves the transfer of the appropriate DNA from the interior of the nucleus to the region of the nuclear periphery and the necessary conformational changes and specific protein interactions that render such DNA susceptible to hydrolysis by DNase I, this in turn make genes also vulnerable to inducers, repressors and other transcriptional

factors. The second step regulates the transition of the exposed genes between active and inactive states as a result of interaction with appropriate effector molecules in the surrounding medium. About 30% of the genes in mammalian cells is converted from sequestered to exposed state in a single action by cAMP, retinoic acid and NGF on transformed cells. Puck and Krystosek using Chinese hamster ovary cells[20] have shown that cytoskeleton network is a necessary structure in producing the specific genome exposure pattern for each cell type. It should also be noted that genome exposure is necessary but not sufficient condition for gene activation.

The mammalian cell cytoskeleton thus becomes part of an information transmission system extending from the cell membrane to its specific receptor sites through the cytoplasm and terminating in specific points on each chromosome so that specific domains of exposure and sequestration result.

Conclusion

The structural and functional integrity of a cell is maintained by the cytoskeletal network, cytoskeletal-associated proteins and the extracellular matrix proteins. Fibronectin, laminin, collagen which are regulated by hormones are connected to the actin cytoskeleton via their transmembrane receptors. The actin network is in turn linked to IFs and MTs. A coordination between these various components is necessary to maintain a definite cell shape in a given physiological condition. For instance, disruption of cytoskeletal-ECM linkage in skeletal muscle leads to sarcolemmal instability, muscle cell necrosis resulting in muscular dystrophy[105]. In experiments with xenopus embryos, introduction of mutant E-cadherins into embryos resulted in ectodermal lesions at the beginning of gastrulation, later causing disruptions in early epidermal development[106]. Expression of mutant N-cadherin (N-neural) lacking the ectodomain resulted in general inhibition of adhesive interaction in the embryo[107]. Similarly disruption of cytokeratin expression during development of xenopus embryo resulted in defective gastrulation. Blessing *et al.*[108,109] have shown that correct IF can be of critical importance for the function and stability of a given cell type.

Hormones affect their target cells during cell growth, division, differentiation, transformation and apoptosis. The mechanism involved in the cell shape change during these processes is still not clear, however, hormones are known to regulate ECM proteins which effect cytoskeletal membrane interaction bringing about altered cell morphology. The cytoskeletal associated proteins help in the polymerization/depolymerization of the cytoskeleton elements. The cytoskeleton network is an active site for signal transduction pathways. The various molecules involved in these pathways are coming to light and a clear-cut pathway involved in hormone-induced cell shape changes is yet to be identified. However, the role of cytoskeleton, their associated proteins and ECM is well established, but how these different components coordinate to maintain a definite cell shape in a given environmental milieu is still an active field of research.

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MEETINGS/SYMPOSIA/SEMINARS

Third National Seminar on Malaria and Other Tropical Diseases

Date: 18–20 February 1997
Place: Bangalore

Topics include: Genetics and cytogenetics of insect vectors, Physiology and biochemistry; Radiation and autoradiography, Ecology and ethology, Insecticide resistance; Speciation and evolution, Chemotherapy and Immunology, Vector control – genetic, biological and chemical. Social and economic research; other related studies (insect pests of plants and animals). Epidemiology of tropical diseases; The influence of genetic and other factors on vector susceptibility to parasites; Genetic and other factors on vector susceptibility to parasites; Genetic engineering technology in vector control.

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Silver Jubilee Symposium of Catalysis Society of India: Thirteenth National Symposium on Catalysis

Date: 2–4 April 1997
Place: Dehradun

This symposium plans to cover all branches of homogeneous and heterogeneous catalysis. Particular emphasis will be given to novel applications in industrial catalysis related to petroleum refining, petrochemicals and fine chemicals. Other areas of interest are catalysis in the protection of environment, C_1 chemistry, microporous materials synthesis and characterization.

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Short Course on Aseismic Design of Structures—Random Vibration Approach

Date: 20–22 May 1997
Place: Bangalore

Course is designed to provide a fundamental understanding of the seismic hazard analysis, theory of random vibrations, and their applications to seismic design of buildings and seismic qualification of secondary structures. It will be useful to: practising engineers or research professionals responsible for ensuring the seismic safety of a structural system or sub-system, other practising engineers requiring the application of random vibration principles (e.g. in the design of vehicular, aerospace and automobile systems), and teachers from engineering colleges.

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