Here, we present evidence that exposure of B-lineage lymphoid cells to low energy electromagnetic fields (EMF) stimulates the protein tyrosine kinases Lyn and Syk, results in tyrosine phosphorylation of multiple electrophoretically distinct substrates, and leads to downstream activation of protein kinase C (PKC). EMF exposure enhances protein tyrosine phosphorylation in Syk deficient but not in Lyn-deficient B-lineage lymphoid cells and stimulates Lyn kinase activity in wild-type as well as Syk-deficient B-lineage lymphoid cells. These results indicate that activation of Lyn kinase is sufficient and mandatory for EMF-induced tyrosine phosphorylation in B-lineage lymphoid cells. The PKC activity increases later than the Lyn activity and pretreatment with the PTK inhibitors genistein or herbimycin A abrogates the EMF-induced PKC signal. Thus, stimulation of Lyn is a proximal and mandatory step in EMF-induced activation of PKC in B-lineage lymphoid cells. Our observations prompt the hypothesis that a delicate growth regulatory balance might be altered in B-lineage lymphoid cells by EMF-induced activation of Lyn.

B-lineage acute lymphoblastic leukemia (ALL)\(^1\) is the most common form of childhood cancer (1, 2). B-lineage ALL is a heterogeneous group of diseases that are thought to originate from putative developmental lesions in normal B-lymphocyte precursor clones during early phases of ontogeny, which allegedly lead to maturational arrest at discrete stages of lymphopoiesis. It is generally believed that such developmental lesions lead to an uncoupling of proliferation and differentiation in target lymphocyte precursor clones (1, 2). The stabilization and subsequent clonal expansion of usually transient B-lymphocyte precursor cell phenotypes have been proposed as critical proximal events in the leukemogenesis of B-lineage ALL (1-3).

Recently, concern has emerged about the possibility that EMF radiation from residentially proximate power lines, household electrical wiring, and appliance usage may contribute to the risk of childhood ALL (4-12). Several studies of associations between childhood leukemia and residential exposure to electric power lines yielded epidemiologic evidence for a link between EMF exposure and ALL (4-13). A recent study by the Swedish National Institute for Occupational Health indicated that children in Sweden chronically exposed to powerline frequency magnetic fields exhibited a 3-fold increase in their incidence of leukemia (14), and similar data have been reported by other investigators (14-20).

The molecular mechanism by which EMF exposure could participate in the complex process leading from changes in cell properties to the actual development of B-lineage ALL in vivo has not been deciphered. No directly genotoxic actions of low energy EMF are known, and it is widely agreed that exposure of cells to nonionizing EMF does not produce mutations or chromosome damage (17–27). Thus, any participation by EMF in leukemogenesis of B-lineage ALL is likely to be by means of influencing survival, proliferation, and/or differentiation of B-lineage lymphoid cells rather than by producing the primary mutational or "initiation" event. A point of exceptional experimental focus in current EMF research is the investigation of EMF effects on signal transduction pathways (28–34). Several investigators noted that EMF exposure activates a protein kinase C (PKC)-linked signaling cascade (34, 35). However, EMF-induced signaling events proximal to PKC activation have not been evaluated.

Protein tyrosine kinases (PTK) participate and play pivotal and myriad roles in initiation of signal cascades that affect proliferation and survival of human B-lineage lymphoid cells (36–43). Notably, recent studies indicate that stimulation of protein tyrosine kinases in B-lymphocyte precursors is a requisite step in the generation of the pleiotropic effects of ionizing radiation, including the activation of protein kinase C (PKC) and PKC-dependent serine kinases, nuclear factor κB (NF-κB), as well as c-jun proto-oncogene expression (36–38). A delicate balance of oncogenic versus tumor-suppressive proteins might be altered when a tyrosine kinase regulatory pathway is activated (44).

The purpose of this study was to examine the role of PTK in EMF-induced activation of the PKC pathway in B-lineage lymphoid cells. Here, we present evidence that stimulation of PTK is an important and mandatory proximal step in EMF-induced activation of the PKC signaling cascade in B-lineage lymphoid cells.

EXPERIMENTAL PROCEDURES

Cell Lines—Nalm-6 human pre-B cells (39) were maintained in suspension cultures in RPMI 1640 supplemented with 2.5% fetal calf...
EMF-induced Lyn Kinase Signal

EMF exposure induces tyrosine phosphorylation of multiple electrophoretically distinct substrates in B-lineage lymphoid cells. NALM-6 human pre-B or DT-40 chicken B cells were exposed to 60-Hz EMF for the indicated periods. After EMF exposure, cells were lysed with SDS lysis buffer and boiled. Equivalent amounts of protein were fractionated on 10% polyacrylamide gels, transferred to Immobilon-P PVDF membranes, and immunoblotted with an anti-phosphotyrosine antibody followed by incubation with 125I-protein A (1 μg/ml) and autoradiography for detection of phosphotyrosyl proteins. Controls included unstimulated samples (CON, negative control) as well as samples stimulated with an anti-CD19 × CD19 monoclonal antibody homogenate (1 μg/ml × 10 min) (39). Molecular masses (in kDa) of the phosphotyrosyl protein substrates were calculated from prestained molecular size markers run as standards.

- Serum, 10 mM L-glutamine, and 5 mM pyruvate. Wild-type and mutant DT-40 cells (45, 46) were maintained in suspension cultures at 37 °C, 5% CO2 in a humidified incubator. The culture medium was RPMI 1640 supplemented with 10% fetal calf serum, 2.5% chicken serum, 10 mM L-glutamine, and 5 mM pyruvate. Wild-type and mutant clonal antibody homoconjugate (1 μg/ml) served as negative control as well as samples stimulated with an anti-CD19 × CD19 monoclonal antibody homogenate (1 μg/ml × 10 min) (39). Molecular masses (in kDa) of the phosphotyrosyl protein substrates were calculated from prestained molecular size markers run as standards.

- PKC Assays—PKC activity was measured by incubating aliquots of cytosolic or membrane fractions with [γ-32P]ATP (3000 Ci/mmol, Amersham) in a solution containing (final concentrations) 20 mM Tris, pH 7.5, 20 mM NaCl, 200 mM CaCl2, 20 μM ATP, 10 μM phorbol 12-myristate 13-acetate, and 0.28 mg/ml phosphatidylserine, with 25 μM concentration of a specific peptide substrate for PKC corresponding to sequence positions 4–14 of myelin basic protein, QKRPSQRSKYL (49), according to published procedures (50). The 5 min in vitro kinase reaction was stopped by spotting aliquots of the reaction mixture (25 μl) onto phosphocellulose paper. The paper disks were washed three times with 1% (v/v) phosphoric acid and counted by liquid scintillation.

RESULTS AND DISCUSSION

EMF Exposure of B-lineage Lymphoid Cells Induces Enhanced Tyrosine Phosphorylation of Multiple Substrates—To examine the effects of EMF on protein tyrosine phosphorylation in B-lineage lymphoid cells, we first compared the profiles of tyrosine-phosphorylated proteins in whole cell lysates of NALM-6 human pre-B cells, prepared at various time points after exposure to low energy EMF (1 Gauss, 60 Hz), by Western blot analysis using a polyclonal anti-phosphotyrosine antibody. Fig. 1 illustrates that EMF exposure of NALM-6 cells results in enhanced tyrosine phosphorylation of constitutively tyrosine-phosphorylated abundant phosphoprotein substrates with apparent molecular masses of 55 kDa, 76 kDa, 95–97 kDa, 120 kDa, 150 kDa, and 190 kDa within 10 min. Tyrosine phosphorylation showed a biphasic course with a slight increase detectable by 1–3 min (first peak) followed by dephosphorylation between 3 and 5 min, and a rapid induction to maximum levels between 5 and 10 min (second peak) followed by gradual dephosphorylation. In addition, a 72-kDa protein substrate with minimal baseline tyrosine phosphorylation became markedly tyrosine-phosphorylated after 10 min of EMF exposure (Fig. 1). Similarly, exposure of DT-40 chicken B-cells to EMF causes enhanced tyrosine phosphorylation of the 55-, 72-, 76-, 95-, 120-, and 150-kDa substrates. However, the kinetics of tyrosine phosphorylation was different from the kinetics observed in NALM-6 cells in that tyrosine phosphorylation was monophasic, and maximum tyrosine phosphorylation was observed after a 1-min EMF exposure.

EMF Exposure of B-lineage Lymphoid Cells Stimulates the Activity of the Src Proto-oncogene Family Tyrosine Kinase Lyn—In B-lineage lymphoid cells, CD19 is physically and functionally associated with Src family protein tyrosine kinases...
EMF-induced Lyn Kinase Signal

**Fig. 2. Time course of Lyn kinase activation after EMF exposure.** A, unstimulated NALM-6 human pre-B cells were lysed in a Nonidet P-40-containing buffer, and equal amounts of lysate (200 μg of protein/reaction mixture) were used for immunoprecipitation and immune complex kinase assays of the indicated Src family PTK, as described under “Experimental Procedures.” No primary antibody was added to the lysate of the control sample (first lane from left). B, NALM-6 cells were exposed to 60-Hz EMF for the times indicated, pelleted, lysed in Nonidet P-40 buffer. Lyn kinase was immunoprecipitated, and immune complex kinase assays (39) were performed using half of the samples, as described under “Experimental Procedures.” Autophosphorylation was quantitated by a 4-min liquid scintillation counting of the incorporated 32P in the excised Lyn kinase bands.

**Fig. 3. Time course of Syk kinase activation after EMF exposure.** A, NALM-6 cells were exposed to 60-Hz EMF for the times indicated, pelleted, and lysed in Nonidet P-40 buffer. Syk kinase was immunoprecipitated and immune complex kinase assays (39) were performed using half of the samples, as described under “Experimental Procedures.” Autophosphorylation was quantitated by a 4-min liquid scintillation counting of the incorporated 32P in the excised Syk kinase bands. B, the other half of the Syk immunoprecipitates were fractionated first on 9.5% polyacrylamide gels, electrophoretically transferred to Immobilon-polyvinylidene difluoride membranes and immunoblotted for 1½ h with anti-Syk antibody (1:100 dilution) followed by incubation with 125I-protein A (specific activity = 30 μCi/μg; ICN Biomedicals) and autoradiography for detection of Syk.

The engagement of the CD19 receptor with an anti-CD19 monoclonal antibody homoconjugate rapidly activates the associated PTK and results in tyrosine phosphorylation of multiple substrates (39). The profile of anti-phosphotyrosine reactive protein substrates generated by EMF exposure of NALM-6 cells was essentially identical with that generated by the engagement of the CD19 receptor with an anti-CD19 monoclonal antibody homoconjugate (Fig. 1, left panel). This intimated the possibility that EMF exposure also effects the activation of Src family PTK. To examine whether this might be the case, NALM-6 cells were exposed to EMF (1Gauss, 60 Hz), and the PTK activity of Lyn, which is the predominant Src family tyrosine kinase in NALM-6 cells (see Fig. 2A), was estimated by immune complex protein kinase assays measuring autophosphorylation. As shown in Fig. 2B, EMF exposure lead to rapid stimulation of Lyn kinase activity in NALM-6 cells. This stimulation was evident as early as 30 s after EMF exposure and reached a maximum at 1 min, representing a 4.8-fold increase in PTK activity (i.e. 99,283 cpm/107 cells versus 20,898 cpm/107 cells). The comparison of the Lyn kinase activities in cells cultured overnight in the absence of EMF versus in the presence of continuous EMF exposure showed a >9-fold higher activity in EMF-treated cells (6,127 cpm versus 55,603 cpm, see the last two lanes of Fig. 2B). The abundance of the enzyme, as estimated by anti-Lyn immunoblotting, did not change during the course of the experiment suggesting altered specific activity (Fig. 2C). Similar results were obtained with NALM-6-UM1 cell line, a NALM-6 subclone which causes disseminated and fatal leukemia when injected into SCID mice (51) as well as the DT-40 chicken B-cell line (data not shown). These results demonstrate that activation of Src family PTK is an integral component of the signal transduction cascade triggered in response to EMF exposure.

**EMF Exposure of B-lineage Lymphoid Cells Stimulates the Activity of Syk Kinase—** Syk kinase is a 72-kDa non-receptor PTK which is involved in important physiologic signaling events in B-lineage lymphoid cells, including the B-cell receptor-coupled calcium mobilization (45, 46, 52–54). Syk kinase can be activated by several members of the Src PTK family (46). Sidorenka et al. (55) demonstrated that Syk kinase associates with Lyn kinase in B-lineage lymphoid cells. Syk kinase has also been shown to mediate inositol trisphosphate generation in chicken B-lineage lymphoid cells, and Syk-deficient B-cells lack tyrosine phosphorylation of PLC-γ2 resulting in the loss of calcium mobilization upon antigen receptor stimulation (45). Therefore, we were compelled to study the effects of EMF exposure on the enzymatic activity of Syk kinase in B-lineage lymphoid cells. As shown in Fig. 3A, EMF exposure of NALM-6 human pre-B cells resulted in Syk kinase activation within 30 s, and maximum stimulation was observed at 1 min followed by rapid deactivation. At 20–30 min after EMF exposure, the Syk...
kinase activity was lower than at baseline (9–10 × 10^3 cpm versus 24 × 10^3 cpm; Fig. 3A). The enzymatic activity of Syk returned to baseline at 1 h. The abundance of the enzyme, as estimated by anti-Syk immunoblotting, did not change during the course of the experiment suggesting altered specific activity (Fig. 3B).

**EMF-induced Activation of Lyn Kinase Does Not Depend on Syk Kinase and Lyn Kinase Is Essential for EMF-induced Activation of Syk Kinase**—In NALM-6 cells, Syk kinase activation was not as pronounced as Lyn kinase activation and it lasted less than 3 min, whereas Lyn kinase activation lasted at least 1 h (see Figs. 2 and 3). These results implicated Lyn kinase in the induction of EMF-dependent tyrosine phosphorylation in B-lineage lymphoid cells. To directly evaluate the relative contributions of Lyn and Syk kinases to EMF-induced tyrosine phosphorylation, we used the DT-40 model system (45, 46).

DT-40 is a chicken B-cell line that is immunologically very similar to human B-lineage lymphoid progenitor cells (45, 46). Like human B-lineage lymphoid cells that are thought to be the progenitors of leukemic cells in children with B-lineage ALL, DT-40 cells express high levels of Lyn and Syk tyrosine kinases but not the other members of the Src- or Syk-PTK families (see Refs. 45 and 46 and Fig. 4A). Lyn knock-out and Syk knock-out mutants of the DT-40 cell line were established by targeted gene disruption (see Ref. 45 and Fig. 4A). Defects of early signaling events are different between Lyn-deficient and Syk-deficient mutants, demonstrating that Lyn and Syk kinases mediate discrete signaling functions through their enzymatic activities (45). As shown in Fig. 4B, EMF stimulated Lyn kinase activity in wild-type as well as Syk-deficient DT-40 cells. Furthermore, EMF exposure enhanced protein tyrosine phosphorylation in Syk-deficient but not in Lyn-deficient DT-40 cells (data not shown). Our results indicate that activation of Lyn kinase is sufficient and mandatory for EMF-induced tyrosine phosphorylation in DT-40 cells. However, these experiments in the DT-40 model system failed to provide conclusive information regarding the role of Lyn kinase in EMF-induced activation of Syk kinase because no significant Syk kinase stimulation was observed in wild-type DT-40 cells or Lyn-deficient DT-40 cells (data not shown).

**Role of Tyrosine Phosphorylation in EMF-induced Activation of PKC in B-lineage Lymphoid Cells**—Recent studies have demonstrated that stimulation of Src family PTK in B-lineage lymphoid cells is a requisite and proximal step in the activation of PKC and PTK-linked distal signaling events, including activation of nuclear factor κB and c-jun proto-oncogene expression after engagement of functionally important membrane receptors (39), ionizing radiation (37, 38), and UV radiation (56). Therefore, we next examined the effects of tyrosine kinase inhibitors genistein and herbimycin on EMF-induced PKC activation in NALM-6 cells. As shown in Fig. 5, there was a marked EMF-induced increase in membrane-associated PKC activity which started at 5 min and continued for 60 min. Thus, PKC activity was stimulated much later than the activity of Lyn or Syk tyrosine kinases (see Fig. 2), suggesting that it may be triggered by tyrosine phosphorylation. Notably, treatment with herbimycin A (1 μg/ml × 18 h) or genistein (30 μg/ml × 30 min) prior to EMF exposure abrogated the EMF-induced PKC activation, providing evidence that stimulation of Src family PTK is an important and proximal step in EMF-induced activation of PKC in B-lineage lymphoid cells. These results are reminiscent of the role of tyrosine kinases in γ-ray- or B43(anti-CD19) antibody-induced activation of PKC in B-lineage lymphoid cells (37–39).

In summary, we examined the biochemical nature of signaling events in B-lineage lymphoid cells exposed to low energy EMF. This report clarifies the chronological sequence of biochemical events that follow EMF exposure and provides unprecedented evidence that EMF exposure initiates a signaling cascade that is intimately linked to the Src proto-oncogene family PTK Lyn. Lyn kinase participates and plays pivotal and
EMF-induced Lyn Kinase Signal

myriad roles in initiation of signal cascades that affect proliferation and survival of B-lineage lymphoid cells (37–39). Our observations prompt the hypothesis that a delicate growth-regulatory balance might be altered in B-lineage lymphoid cells by EMF-induced activation of Lyn.

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Exposure of B-lineage Lymphoid Cells to Low Energy Electromagnetic Fields Stimulates Lyn Kinase

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J. Biol. Chem. 1995, 270:27666-27670.
doi: 10.1074/jbc.270.46.27666

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