We have isolated cDNA encoding human telencephalin (TLN), a brain segment-specific neuronal adhesion molecule. Human TLN comprises an NH₂-terminal signal peptide, an extracellular region with nine Ig-like domains, a single transmembrane region, and a COOH-terminal cytoplasmic tail. The NH₂-terminal five Ig-like domains of TLN were closely related to those of intercellular adhesion molecules (ICAMs)-1 and -3. The TLN gene was mapped to the human chromosome 19p13.2, where the ICAM-1, -3, and -4 (LW) genes are located. Furthermore, we observed lymphocyte function-associated antigen-1 (LFA-1)-mediated adhesion of HL-60 cells on recombinant TLN protein, as well as on ICAM-1. However, the interaction of TLN with LFA-1 on HL-60 cells was divalent cation-independent and phorbol 12-myristate 13-acetate stimulation-independent. We conclude that TLN is a unique neuronal member of ICAM sub-group of the Ig superfamily and propose a novel type of interaction between the Ig superfamily molecule and integrin, which does not require the activation of integrin. TLN on the surface of telencephalic neurons may be a target molecule in the brain for LFA-1-expressing microglia or leukocytes in physiological or pathological conditions.

The most conspicuous feature of the human brain lies in its highly developed, enormously enlarged, and elaborated telencephalon (1). The telencephalon is the most rostral brain segment, which includes the cerebral neocortex, olfactory cortex, hippocampus, striatum, amygdala, septum, and olfactory bulb. These telencephalic regions take charge of higher brain functions such as memory, learning, emotion, sensory perception, and voluntary movements.

Telencephalin (TLN)1 is a cell surface glycoprotein whose expression is confined exclusively within the telencephalon (2–4). TLN is expressed by subsets of the telencephalic neurons, but not by glial cells. In the neurons, TLN is localized to soma-dendritic membrane, but not to axonal membrane. In the course of brain development, TLN first appears around birth when the dendritic outgrowth and branching, spine formation, and synapse formation occur in the telencephalic regions. Afterwards, TLN expression persists even in adult animals. These unique expression patterns of TLN in brain segment-, neuronal subsets-, soma-dendritic membrane-, and developmental stage-specific fashions suggest that TLN may be a crucial cell surface molecule for the formation, maintenance, and plasticity of neuronal networks in the brain (2–7).

We previously cloned cDNAs for rabbit and mouse TLN and demonstrated that TLN is a type I integral membrane protein belonging to the immunoglobulin (Ig) superfamily (7). Although there exist several members of the Ig superfamily that are expressed in brain region- and neuronal type-specific manners, such as limbic system-associated membrane protein (LAMP) (8), neurotrimin (9), TAG-1 (10), F3 (11), BIG-1 (12), and BIG-2 (13), TLN shows the most restricted pattern of expression. Of all the Ig superfamily molecules so far identified, the structure of TLN is most closely related to those of intercellular adhesion molecules (ICAMs)-1 and -3. The NH₂-terminal Ig-like domain of TLN contains four cysteine residues that are capable of forming two intradomain disulfide bridges. Similarly spaced cysteine residues in the NH₂-terminal Ig-like domains are seen in a unique subgroup of the Ig superfamily, such as ICAM-1 (14, 15), ICAM-2 (16), ICAM-3 (17, 18), ICAM-4 (LW antigen) (19, 20), vascular cell adhesion molecule-1 (VCAM-1) (21), and mucosal addressin cell adhesion molecule-1 (MadCAM-1) (22). These molecules have been reported to use members of the integrin family as their counter-receptors (20, 23), implicating a possible interaction of TLN with a certain integrin molecule (4).

In this paper, we cloned cDNA for human TLN, mapped its chromosomal locus, and showed an interaction of TLN with lymphocyte function-associated antigen-1 (LFA-1, CD11a/CD18, or αiβ2 integrin), a common counter-receptor for ICAM-1, -2, -3, and -4 (20, 23). The TLN/LFA-1 binding provides a basis for understanding molecular mechanisms underlying cell-cell interactions between telencephalic neurons and LFA-1-expressing microglia or leukocytes.
EXPERIMENTAL PROCEDURES

cDNA Cloning of Human TLN—We used a PCR-based strategy for cDNA cloning of human TLN. Two degenerate oligonucleotides (5'-acigti(ac)gigtigc(acgt)gg(acgt)cc(acgt)tggct(acgt)tgg-3' and 5'-ccic-cigcicc(ag)tt(acgt)ag(ag)ca(acgt)ac(acgt)gc(ct)tc-3') corresponding to the amino acid sequences TVRVAGPWLW and EAVCLNGAGG (Fig. 1, dashed underlines) that are completely conserved between mouse and rabbit TLN (7) were synthesized and used as primers in PCR with human cerebral neocortex cDNA as a template. PCR was performed using Taq DNA polymerase (Toyobo, Japan) under the following conditions: 96°C for 1 min, 55°C for 2 min, 72°C for 2 min for 40 cycles, then 72°C for 3 min. The PCR product (186 base pairs) was directly cloned into pCRII vector (Invitrogen, Netherlands) and sequenced by the dideoxy chain termination method (24) using Sequenase kit (U.S. Biochemical Corp., Amersham, United Kingdom).

To obtain full-length cDNA for human TLN, an unamplified human cerebral neocortex cDNA library (9 x 10⁵ recombinant phages) was screened with the 32P-labeled PCR product (186 base pairs) as a probe. Twelve clones were randomly selected from the 50 positives of the first screening, isolated, and subcloned into the EcoRI site of pBlueScript SK(+) (Stratagene, La Jolla, CA). Restriction mapping and partial sequencing revealed that all 12 clones were derived from the same gene. The complete DNA sequence was determined for both strands of the longest clone hTLN-#12.

Northern Analysis—Equal amounts of total RNA from human hippocampus and cerebellum were size-fractionated in 1.5% formaldehyde-agarose gel and transferred to nylon membrane (Hybond N, Amersham, United Kingdom). An Apo1 fragment (828 base pairs, residues 507-1334) of human TLN cDNA was 32P-labeled with a random priming kit (Boehringer Mannheim) and used as a probe. Hybridization, washing, and visualization were performed as described previously (7).

Chromosomal Mapping by Fluorescence in Situ Hybridization—Fluorescence in situ hybridization (FISH) was carried out as described (20). In brief, human (pro)mitotic chromosomes were prepared from normal male lymphocytes using the thymidine synchronization, BrdU release technique for the delineation of G-bands. Before hybridization, chromosomes were stained in Hoechst 33258 and irradiated with UV. A 3.0-kilobasepair full-length cDNA of the human TLN was labeled with biotin-16-dUTP by nick translation and hybridized to the denatured chromosome slides at a final concentration of 25 ng/ml in a mixture of 50% formamide, 10% dextran sulfate (Sigma), 2 x SSC, sonicated salmon sperm DNA (2 mg/ml), and E. coli tRNA (2 mg/ml). The hybridization signals were detected with FITC-avidin (Boehringer Mannheim), and chromosomes were counterstained with propidium iodide (1 µg/ml). The precise signal position was determined by the delineation of G-banding patterns.

Production of Recombinant Fc-chimeric Proteins—Recombinant soluble Fc-chimeric proteins were produced essentially as described (27). Briefly, for preparation of human TLN/Fc protein, the mammalian expression plasmid containing the human TLN cDNA encoding the extracellular region (a signal peptide and distal five or nine Ig-like domains) and the human IgG1 gene Fc region (28) in pEF-BOS (29) was transfected into COS7 cells by the standard DEAE-dextran transfection method and purified from culture supernatants using a protein A-Sepharose column (Pharmacia Biotech Inc., Uppsala, Sweden). Human ICAM-1/Fc, rat BIG-1/Fc, rat BIG-2/Fc, and signal peptide/Fc (SP/Fc) were prepared by similar procedures.
Cell Adhesion Assay—Cell adhesion assay was performed essentially as described (30). Immulon-3 96-well plastic plates (Dynatech, Chantilly, VA) were coated with 1 μg/well goat anti-human Fc IgG (Sigma) in phosphate-buffered saline overnight at 4°C, blocked with 0.4% bovine serum albumin in phosphate-buffered saline for 2 h at room temperature, and then incubated with Fc-chimeric proteins in 25 mM Tris-HCl (pH 8.0), 150 mM NaCl, and 2 mM MgCl₂ for at least 2 h at room temperature.

The HL-60 human promyelocytic leukemia cells were grown in RPMI 1640 medium supplemented with 20% fetal bovine serum. Prior to the adhesion assay, the cells were labeled for 12–24 h with [3H]thymidine at 10 μCi/1 × 10⁶ cells, harvested, and washed three times with the assay medium (RPMI 1640 supplemented with 2.5% fetal bovine serum) prewarmed at 37°C. In order to increase avidity of LFA-1, the labeled cells were stimulated with 80 nM phorbol 12-myristate 13-acetate (PMA, Wako, Osaka, Japan) for 30 min at 37°C. The cells were added to the Fc-chimeric protein-coated plates to give 1.5 × 10⁵ cells/well in a final volume of 100 μl and incubated for 30 min at 37°C. Cells that remained bound after three washes were lysed in 1% SDS, and radioactivity was counted using a liquid scintillation analyzer.

In the experiments using blocking antibodies, the PMA-stimulated HL-60 cells were preincubated with 2 μg/ml of antibodies for 30 min at 4°C and then added to the plates. Antibodies used in this study were the anti-CD11a mAbs, MHM-24 (Dako, Glostrup, Denmark), HI111, G43–25B (Pharmingen, San Diego, CA), TP1/32 (Upstate Biotechnologies, Inc., Lake Placid, NY), 38 (Ancell, Bayport, MN); anti-CD11b mAb, 2LPM19c (Dako); anti-CD11c mAb, 3.9 (Sigma); and anti-CD18 mAbs, MHM-23 (Dako), YFC118.3 (Serotec, Oxford, UK).

RESULTS

cDNA Cloning of Human TLN—To isolate cDNA for human TLN, we performed PCR using human cerebral neocortex cDNA template and two degenerate oligonucleotide primers (Fig. 1, dashed underlines) based on the conserved amino acid sequences of mouse and rabbit TLN (7). An appropriately sized PCR product (186 base pairs) was subcloned, sequenced, and used as a probe for the following plaque hybridization. The first
Neuronal Glycoprotein Telencephalin Binds LFA-1 Integrin

The extracellular region of TLN comprises nine tandemly arranged Ig-like domains, rendering TLN to a member of the Ig superfamily (34–36). In particular, the distal eight Ig-like domains are closely related to those of ICAMs (14–20) (Fig. 4). Total amino acid identity is 50% with ICAM-1 (domains I–V), 55% with ICAM-3 (domains I–V), 38% with ICAM-2 (domains I and II), and 32% with ICAM-4 (domains I and II). The highest homology is observed with the domain II of ICAM-1 and the domains II–IV of ICAM-3 with more than 64% amino acid identity. Together with the presence of characteristic four cysteine residues in the first domain, it will be concluded that TLN is a novel member of the ICAM subgroup of the Ig superfamily.

The chromosomal localization of human TLN gene was determined using the FISH technique. The full-length cDNA of the human TLN, which did not cross-hybridize to ICAM-1 and ICAM-3 in genomic Southern blot analysis (data not shown), was used as a probe for the FISH mapping. Among 52 (pro) metaphase chromosome spreads analyzed, 16 showed twin-spot signals on both homologous chromosomes 19p. Since the short arm of chromosome 19p contains mostly R-bands, it is difficult to visualize high-resolution bands by R-banding FISH. Thus, we detected FITC signals on propidium iodide-stained metaphase chromosomes and then the sublocalization was confirmed by delineation of the high-resolution G-bands. In this system, the precise locus of the human TLN gene was localized on 19p13.2 as shown in Fig. 5. Interestingly, the genes of human ICAM-1 (37), ICAM-3 (38), and ICAM-4 (39) were also mapped to this locus, suggesting that these four molecules were derived from a common ancestral gene by gene duplication.

**Adhesion of LFA-1-expressing HL-60 Cells to TLN—**Of all the members of the Ig superfamily, ICAM-1, -2, -3, -4, VCAM-1, and MadCAM-1 are characteristic in that they interact with integrin counter-receptors (20, 23). The amino-terminal Ig-like domains of these six molecules have similarly arranged four cysteine residues, suggesting that formation of two intradomain disulfide bonds might be of functional importance for the interaction with integrins. These four cysteine residues are well conserved also in the first Ig-like domain of TLN, raising a possibility of the interaction of TLN with integrin(s). Furthermore, the highly conserved residues proposed to be a critical part of an integrin-binding structure in the first Ig-like domains of ICAMs and VCAM-1 (30, 40–43) are also found in TLN (boxed region in Fig. 6). In particular, human TLN has an identical amino acid sequence in this region with human ICAM-3 and mouse ICAM-2, suggesting that TLN might interact with LFA-1 integrin, a common counter-receptor for ICAMs. To examine this possibility, we performed an adhesion assay with LFA-1-expressing HL-60 cells and purified TLN protein.

Soluble recombinant fusion proteins were produced and purified to nearly homogeneity (Fig. 7). TLN(1–5)/Fc comprises the amino-terminal five Ig-like domains of human TLN and theFcregion of human IgG1. ICAM-1/Fc, a positive control, contains all the five Ig-like domains of human ICAM-1 fused to the Fc. Signal peptide/Fc (SP/Fc), a negative control, contains the ICAM-1 signal peptide and the Fc. Cell adhesion assays were carried out with the chimeric proteins immobilized onto the plastic plates via anti-human IgG antibody.

When PMA-stimulated HL-60 cells were added into the Fc-chimeric protein-coated wells and incubated at 37 °C for 30 min, about 40% of the added cells bound strongly onto theTLN(1–5)/Fc-coated wells (Fig. 8B). In the same condition, about 60% of the cells bound onto the ICAM-1/Fc-coated wells (Fig. 8C). In contrast, only 5% bound nonspecifically to SP/Fc (Fig. 8A) or bovine serum albumin (data not shown), suggesting...
that the Fc portion is not responsible for the adhesion. The HL-60 adhesion was dependent on the amount of coated proteins (Fig. 9). A similar result of the HL-60 adhesion was obtained when TLN(1–9)/Fc, a fusion protein consisting of the whole extracellular region of TLN and the IgG1 Fc region, was used as a substrate (data not shown). Fc-chimeric proteins of the other two neuronal Ig superfamily molecules, BIG-1 (12) and BIG-2 (13), did not support the adhesion of HL-60 cells (data not shown), indicating that the binding of HL-60 cells to TLN is specific.

To clarify whether leukocyte integrins are involved in this distinctive adhesion of HL-60 cells to TLN/Fc, a panel of different mAbs were tested for adhesion-blocking activity (Fig. 10). Of the seven mAbs against LFA-1, two mAbs against CD11a (HI-111 and MHM-24) and a mAb against CD18 (MHM-23) selectively inhibited the HL-60 adhesion to ICAM-1/Fc, but not to TLN/Fc, while one mAb against CD11a (G43–25B) showed a reciprocal profile with selective and complete blockade of the HL-60/TLN adhesion. Two anti-CD11a mAbs (TP1/32, 38) and one anti-CD18 mAb (YFC118.3) significantly blocked the adhesion to both ICAM-1/Fc and TLN(1–5)/Fc. The HL-60 adhesion to both ICAM-1/Fc and TLN(1–5)/Fc was not affected by mAbs against CD11b (2LPM19c) and CD11c (3.9) (data not shown). These results suggest that the LFA-1 integrin (CD11a/CD18) is involved in the HL-60 adhesion to TLN(1–5)/Fc and that overlapping but different portions of the LFA-1 molecule interact with TLN and ICAM-1.

It has been reported that most of the interaction mediated by integrins are dependent on the presence of divalent cations in the extracellular milieu and that the activation of integrin-expressing cells induce conformational change of the integrins from a low- to high-avidity form (44–46). These two properties hold true for the binding between ICAM-1 and LFA-1. As shown in Fig. 11, the addition of EDTA completely abolished the HL-60 adhesion to ICAM-1/Fc, and the pretreatment of HL-60 cells with PMA resulted in marked enhancement of binding to ICAM-1/Fc. On the other hand, the HL-60 cells behaved in a different manner with regard to the adhesion to TLN/Fc. First, EDTA hardly influenced the HL-60 adhesion to TLN/Fc, indicating no requirement of divalent cations for the binding between TLN and LFA-1. Second, the pretreatment with PMA did not increase the HL-60 adhesion to TLN/Fc,
indicating that the binding between LFA-1 and TLN is activation-independent. These results suggest that LFA-1 on HL-60 cells is constitutively activated for TLN binding, but not for ICAM-1, or that TLN can bind to both resting (low-avidity) and active (high-avidity) forms of LFA-1 on HL-60 cells.

**DISCUSSION**

We have identified and characterized the human TLN. TLN is a type I integral membrane protein with nine Ig-like domains in its extracellular region, belonging to the Ig superfamily. Here, we provided three characteristic features of TLN that indicate an intimate relationship of TLN with ICAMs. First, among all the Ig superfamily molecules so far identified, the Ig-like domains of TLN are most closely related to those of ICAM-1 and ICAM-3 and also show a weaker but significant homology to those of ICAM-2 and ICAM-4. Second, chromosomal locus of the human TLN gene was mapped to 19p13.2 in the vicinity of other three ICAM genes (ICAM-1, -3, and -4). Third, TLN protein was able to bind LFA-1, a common counter-receptor for ICAMs, although the manner of interaction was distinctive (see below). These findings indicate that TLN is a member of ICAM subgroup of the Ig superfamily and shares many properties with hitherto known ICAM subgroup members. However, the expression of TLN is confined to the telencephalon of the central nervous system (2, 3, 7), whereas all the other ICAM members are expressed mostly by cells in the immune and blood systems, such as lymphocytes (ICAM-1 and -3), endothelial cells (ICAM-1 and -2), epithelial cells (ICAM-1), and erythrocytes (ICAM-4) (14–19). Thus, although the molec-
 distint properties of HL-60 cell adhesion to TLN(1–5)/Fc and ICAM-1/Fc. HL-60 cells were added to wells precoated with TLN(1–5)/Fc or ICAM-1/Fc in the presence or absence of 5 mM EDTA. Note that the binding of HL-60 cells onto TLN(1–5)/Fc is divalent cation-independent and PMA-stimulation-independent. Values are represented as means of three to four individual determinations ± S.E.

**Figure 10.** Effects of anti-LFA-1 mAbs on HL-60 cell adhesion to TLN(1–5)/Fc and ICAM-1/Fc. PMA-stimulated HL-60 cells were allowed to bind to ICAM-1/Fc (A) or TLN(1–5)/Fc (B) coated microtiter wells in the presence or absence of mAbs specific for LFA-1. mAbs used were anti-αL subunit (HI-111, MHM-24, TP1/32, 58, G43–25B) and anti-β2 subunit (MHM-23 and YFC118.3). Values are represented as means of three to seven individual determinations ± S.E.

**Figure 11.** Comparison of LFA-1 binding properties of ICAM-1, -2, -3, and TLN.

**Table I**

| ICAM-1 | ICAM-2 | ICAM-3 | TLN |
|--------|--------|--------|-----|
| Requirement for divalent cations | Yes | Yes<sup>a</sup> | Yes<sup>a</sup> | No<sup>b</sup> |
| Activation by phorbol ester | Yes | No<sup>c</sup> | Yes<sup>c</sup> | No<sup>c</sup> |
| Activation by anti-CD44 mAb | Yes<sup>c</sup> | No<sup>d</sup> | No<sup>d</sup> | ND<sup>d</sup> |

<sup>a</sup> Data from Staunton et al. (16) using purified LFA-1.
<sup>b</sup> In the case of LFA-1 on HL-60 cells.
<sup>c</sup> Data from Vermot-Desroches et al. (59) using human T cells.
<sup>d</sup> Not determined.

The adhesion of LFA-1 on the HL-60 cells to TLN/Fc was inhibited by pretreatment of HL-60 cells with two mAbs against CD11a (G43–25B, 38) and one mAb against CD18 (YFC118.3). This result indicates that LFA-1 is a candidate counter-receptor for TLN.

In contrast, three mAbs (HI-111, MHM-24, MHM-23) that completely blocked the LFA-1/ICAM-1 binding had no effect on the binding of HL-60 cells to TLN/Fc. Conversely, a mAb G43–25B strongly inhibited the LFA-1/TLN binding, whereas it showed little effect on the LFA-1/ICAM-1 binding. The overlapping but different profile of these mAbs in adhesion blocking activity indicates that the binding site on LFA-1 for TLN may be close to but distinct from that for ICAM-1. Similar differential effects of a panel of anti-LFA-1 mAbs were reported in T cell binding to ICAM-1 versus ICAM-3 (56). Thus, LFA-1 is capable of binding its multiple counter-receptors in a selective manner.

The binding of LFA-1 on the HL-60 cells to TLN/Fc was unusual in that it required neither divalent cations nor PMA stimulation. Most of the interactions mediated by integrins are dependent on divalent cations and are activated by inside-out signaling (44, 45) with a few exceptions (57, 58). In the case of LFA-1 integrin, four different counter-receptors act in distinctive manners in respect of requirement for divalent cations, activation by phorbol ester, and activation by anti-CD44 antibody (Table I) (59). Our result suggests that LFA-1 on the HL-60 cells may be constitutively activated for the binding to TLN, but not to ICAM-1. A similar activation-independent
binding has been reported in the case of interaction between ICAM-3 and a novel leukointegrin, α6β1 (58).

In the central nervous system, LFA-1 is constitutively expressed by microglia, but not by neurons, astrocytes, nor oligodendrocytes (60, 61). The microglia are also called brain-type macrophages, usually ramified in a resting state, but activated in neurological diseases including Alzheimer’s disease, acquired immune deficiency syndrome, and brain trauma. When activated, the microglia change their shape, migrate to the damaged neurons, proliferate, show neurotoxic activity, and remove dead cells by phagocytosis (62, 63). The constitutive expression of TLN in neurons and LFA-1 in microglia and the present finding demonstrating TLN/LFA-1 binding without intracellular activation suggest that TLN and LFA-1 may mediate neuron/microglia interaction in the telencephalon of the normal brain. This interaction might be necessary for the central nervous system to hold microglia on a tight leash, suppressing its phagocytic and cytotoxic activity in the healthy brain. Alternately, TLN might be a neuronal target molecule for LFA-1-expressing leukocytes and microglia. Such knowledge could help to investigate possible molecular tools that prevent LFA-1-expressing leukocytes and activated microglia from attacking and destroying telencephalic neurons in neurological diseases.

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