Classic Hodgkin's lymphoma (HL) tissue contains a small population of morphologically distinct malignant cells called Hodgkin Reed-Sternberg (HRS) cells, associated with the development of HL. Using 3'-rapid amplification of cDNA ends (RACE) we identified an alternative mRNA for the DEC-205 multiligand receptor in the HRS cell line L428. Sequence analysis revealed that the mRNA encodes a fusion protein between DEC-205 and a novel C-type lectin DCL-1. Although the 7.5-kb DEC-205 and 4.2-kb DCL-1 mRNA were expressed independently in myeloid and B lymphoid cell lines, the DEC-205/DCL-1 fusion mRNA (9.5 kb) predominated in the HRS cell lines (L428, KM-H2, and HDLM-2). The DEC-205 and DCL-1 genes comprising 35 and 6 exons, respectively, are juxtaposed on chromosome band 2q24 and separated by only 5.4 kb. We determined the DCL-1 transcription initiation site within the intervening sequence by 5'-RACE, confirming that DCL-1 is an independent gene. Two DEC-205/DCL-1 fusion mRNA variants may result from cotranscription of DEC-205 and DCL-1, followed by splicing DEC-205 exon 35 or 34–35 along with DCL-1 exon 1. The resulting reading frames encode the DEC-205 ectodomain plus the DCL-1 ectodomain, the transmembrane, and the cytoplasmic domain. Using DCL-1 cytoplasmic domain-specific polyclonal and DEC-205 monoclonal antibodies for immunoprecipitation/Western blot analysis, we showed that the fusion mRNA is translated into a DEC-205/DCL-1 fusion protein, expressed in the HRS cell lines. These results imply an unusual transcriptional control mechanism in HRS cells, which cotranscribe an mRNA containing DEC-205 and DCL-1 prior to generating the intergenically spliced mRNA to produce a DEC-205/DCL-1 fusion protein.

Hodgkin’s Lymphoma Cell Lines Express a Fusion Protein Encoded by Intergenerically Spliced mRNA for the Multiligand Receptor DEC-205 (CD205) and a Novel C-type Lectin Receptor DCL-1*

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The nucleotide sequence(s) reported in this paper has been submitted to the GenBank™/EBI Data Bank with accession number(s) AY184222, AY14006, and AY14007.
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†The abbreviations used are: HL, Hodgkin’s lymphoma; HRS, Hodgkin and Reed-Sternberg; APC, antigen-presenting cell; DC, dendritic cell; RACE, rapid amplification of cDNA ends; RT, reverse transcriptase; CRD, carbohydrate recognition domain; TM, transmembrane domain; CP, cytoplasmic domain; HRP, horseradish peroxidase; mAb, monoclonal antibody; MMR, macrophage mannose receptor; PLA2, phospholipase A2 receptor; ELISA, enzyme-linked immunosorbent assay; PBS, phosphate-buffered saline; SP, signal peptide.
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| Primer | Accession number | Position (orientation) |
|--------|------------------|------------------------|
| 061    | AATCTGCGCGTTTTCGCCAGTGCT | 286–306 (reverse) |
| 062    | GACCATGAGCCCGGCACTGATA | 216–236 (forward) |
| 063    | GGGCCTACACTGCTGTTTGGT | 1811–1831 (forward) |
| 078    | GAAATGTTGAGTACAGAGAGA | 4200–4222 (forward) |
| 086    | ACCAATCACTGCGCCCATGAGA | 5095–5118 (reverse) |
| 087    | ATCGCTGCCGCTGATGCTGACGA | 212–235 (reverse) |
| 088    | TATCAGAGTAAATGACCAGCA | 3327–3347 (forward) |
| 090    | CCAAGGGGCGTACTTCACAAAAA | 2430–2450 (forward) |
| 092    | AGAGAGAACATGATGACAGCA | 1518–1538 (forward) |
| 094    | GAAGACCGTTGAGAAGTATAT | 680–710 (forward) |
| 199    | GATCTGATCCGCTGACTGACGA | 206–226 (reverse) |

(KIAA0022) of DCL-1 was identified by random sequencing of a KG-1 cDNA library (22). Here, we describe the characterization of the DEC-205/DCL-1 fusion mRNA and protein. Its apparently selective expression in HRS cells may make it a useful target for both antibody- and T cell-mediated immunotherapy.

**Experimental Procedures**

**Cell Lines**—The human hematopoietic cell lines, HEL, KG-1, K562, THP-1, U937, Mann, Daudi, Raji, WT49, Mann, Molt-4, Jurkat, HL-60, and HSB-2 were obtained from the American Type Culture Collection (Rockville, MD). L428 cells were provided by V. Diehl (Klinik fur Innere Medizin, Cologne, Germany) (23). HDLM-2 (24) and KM-H2 cells (25) (Rockville, MD). L428 cells were provided by V. Diehl (Klinik fur Innere Medizin, Cologne, Germany) (23). HDLM-2 (24) and KM-H2 cells (25) were obtained from the German Collection of Microorganisms and Cell Culture (Braunschweig, Germany). Mono Mac 6 cells (26) were provided by E. M. Schneider (Dusseldorf, Germany). All cell lines were maintained in RPMI 1640 (Invitrogen, Melbourne, Victoria, Australia), 10% (v/v) fetal calf serum (Invitrogen), 100 units/ml penicillin, and 100 μg/ml streptomycin, except for HSB-2 cells, which were maintained in 20% (v/v) fetal calf serum. These cells were subjected to RNA preparation using TRIzol (Invitrogen) for RT-PCR and Northern blot analysis.

**Antibodies and Other Reagents**—The mAb MMRI-7 against human DEC-205 was produced in our laboratory (27). MMRI-7 binds to an epitope within HRS cells may make it a useful target for both antibody- and T cell-mediated immunotherapy.

**Northern Blot Analysis**—Approximately 10 μg of total RNA from cultured cell lines was fractionated in formaldehyde-denatured 1% (w/v) agarose gel and transferred to a Hybond N+ nitrocellulose membrane (Amersham Biosciences, Sydney, NSW, Australia). The 864-bp DEC-205 cDNA probe nested within DEC-205 CRD1 and -2 was PCR-amplified using primers 094 and 095 on the DEC-205 cDNA clone pCRD1/2-lg (27) and Taq polymerase (Roche Applied Science). The 1617-bp DCL-1 cDNA probe was PCR-amplified using DCL-1-specific primers 062 and 063 on the pBS00-1 (Fig. 1). These probes were purified using a QiAquick PCR purification kit (Qiagen, Clifton Hill, Victoria, Australia) and labeled with [α-32P]dATP (Amersham Biosciences) using a Strip-EZ DNA StipAble DNA probe Synthesis and Removal kit (Ambion, Austin, TX). The membrane was hybridized sequentially with these probes and exposed to a Kodak BioMax MX x-ray film at -70°C using an intensifying screen (Amersham Biosciences). The final wash was 0.1 × SSC (1 × SSC is 0.15 M NaCl, 15 mM sodium citrate, pH 7.0) and 0.5% (w/v) SDS at 68°C. After each probing, the membrane was chemically stripped according to the manufacturer’s instructions and used for hybridization with the other probes.

**5′-RACE**—RNA ligase-mediated 5′-RACE was performed using a FirstChoice RLM-RACE kit (Ambion). Briefly, total RNA from HL-60 was treated sequentially with calf intestinal alkaline phosphatase and tobacco acid pyrophosphatase to select and to remove the cap structure of full-length mRNA. The RNA adaptor was ligated to the RNA using T4 RNA ligase, and the RNA was subjected to cDNA synthesis with random decaer or DCL-1-specific primer 061 and Thermoscript reverse transcriptase (Invitrogen). The cDNA was subjected to two rounds of PCR using DCL-1-specific primers 086 and 099 in combination with the 5′-RACE outer primer and inner primer (provided by the kit), respectively. The PCR product was cloned into pGEM-T Easy vector (Promega) and sequenced.

**Preparation of Cell Lysate**—Approximately 106 cells were lysed with 1 ml of 0.15 M NaCl, 25 mM Tris-HCl, pH 7.4, 1% (v/v) Triton X-100, 0.5% (w/v) sodium deoxycholate, 0.1% (w/v) SDS, and a mixture of protease inhibitors (Complete, EDTA-free, Roche Applied Science) and incubated on ice for 10 min with occasional vortexing. After centrifugation at 12,000 × g for 20 min at 4°C, the supernatant was collected and used directly for immunoprecipitation/Western blotting or sandwich ELISA analysis described below.

**Immunoprecipitation/Western Blot Analysis**—The cell extract was...
precleared with a non-immune rabbit serum and protein A-Sepharose (Sigma) for 1 h at 4 °C and subjected to immunoprecipitation using the rabbit peptide antisera against DEC-205 CP or DCL-1 CP with protein A-Sepharose overnight at 4 °C. The beads were washed with a wash buffer (0.15 M NaCl, 25 mM Tris-HCl, pH 7.5, 0.2% (v/v) Triton X-100, and 0.5% (w/v) sodium deoxycholate), and eluted with SDS-PAGE sample buffer (2% (w/v) SDS, 62.5 mM Tris-HCl, pH 6.8, 0.01% (w/v) bromophenol blue, and 10% (v/v) glycerol) by heating at 95 °C for 5 min. The samples were subjected to Laemmli discontinuous SDS-PAGE with 10% (v/v) polyacrylamide separating gel (28) in the non-reducing condition and transferred to a polyvinylidene fluoride membrane (PVDF-Plus, Osmonics, Westborough, MA). The membrane was blocked with 5% (w/v) nonfat dry milk in PBS/Tween (BLOTTO). To the plate a mixture of DEC-205 mAbs (MMRI-7 and M335, 5 μg/ml each) overnight at 4 °C, and washed with PBS/Tween. The membrane was incubated with HRP-conjugated goat anti-rabbit IgG, and the bound enzyme was detected with enhanced chemiluminescence (SuperSignal West Pico, Pierce, Rockford, IL) on a Kodak X-Omat XB-1 x-ray film.

Sandwich ELISA—An ELISA plate was coated with 10 μg/ml goat anti-mouse IgG in PBS, washed with PBS/Tween, and blocked with BLOTTO. To the plate a mixture of DEC-205 mAbs (MMRI-7 and M335, 5 μg/ml each) was added and incubated for 1 h at room temperature. The plate was washed and incubated with the serially diluted cell extracts overnight at 4 °C. The plate was washed with PBS/Tween and incubated with either rabbit peptide antibodies against DEC-205 CP or DCL-1 CP (1:1000 dilution in PBS/Tween) or non-immune rabbit serum for 1 h at room temperature, and, after washing with PBS/Tween, the plate was incubated with HRP-conjugated goat anti-rabbit IgG in 5% mouse serum and PBS/Tween. The plate was developed with o-phenylenediamine dihydrochloride and quantitated at 492 nm.

RESULTS

Identification of the cDNA Clone Encoding DEC-205/DCL-1 Fusion—To obtain the 3’-end of human DEC-205 mRNA, we performed 3’-RACE (17). This resulted in amplification of a PCR product of ~3 kb (data not shown). When we cloned the PCR product and analyzed several clones by restriction enzyme analysis, however, we realized that there were two distinct sequences within the PCR product. The clone pB30-3 contained the authentic DEC-205 sequence encoding the DEC-205 CRD (Fig. 2). The clone pB30-1, however, encoded DEC-205 CRD 8–10, TM, and CP (17). The other clone pB30-1, however, encoded DEC-205 CRD 8–10 followed by a unique sequence distinct from the DEC-205 TM and CP sequence (Fig. 1A). The junction of the DEC-205 and unique sequence was located within the connecting region (spacer 11) between the DEC-205 CRD10 and TM. A BLAST search identified the unique sequence as a part of the cDNA, KIAA0022 derived from KG-1 cell cDNA library (22). Our further analysis showed that the KIAA0022 contained a partial cDNA encoding a novel type I transmembrane C-type lectin receptor, and we termed it DCL-1 (DEC-205-associated C-type Lectin-1). The complete DCL-1 coding region encodes a signal peptide (SP), one CRD, one TM, and one CP. The KIAA0022 cDNA was recently annotated to a C-type lectin molecule (GenBank™ accession number BA033498), and its gene was mapped to chromosome band 2q24. More details of DCL-1 will be published elsewhere.2

The sequence analysis of the clone pB30-1 showed that fusion junction occurred within the codon G/GC (7’ indicates the junction) for Gly in the DEC-205 spacer 11, connected to the codon G/AC for Asp in the junction between the DCL-1 SP and CRD. The fusion junction was in-frame, connecting the DEC-205 CRD 10 to the DCL-1 CRD, TM, and CP, suggesting that the DEC-205/DCL-1 fusion mRNA is translated. Furthermore, analysis of the DEC-205 and DCL-1 genes indicated that for this fusion mRNA the junction is formed by splicing DEC-205 exon 35 and DCL-1 exon 1, resulting in the fusion of DEC-205 exon 34 to DCL-1 exon 2 (a variant fusion mRNA termed V34-2, 2 S. Khan, K. J. McDonald, B. P. O’Neill, N. Gonzalez, B. J. Cooper, D. N. J. Hart, and M. Kato, manuscript in preparation.

Fig. 1B). An additional variant fusion mRNA termed V33-2 is described below.

The DEC-205/DCL-1 Fusion mRNA Appears to Encode the Entire DEC-205 Ectodomain—We examined the L428 cDNA pool containing the DEC-205/DCL-1 junction by RT-PCR to examine whether it included the entire DEC-205 ectodomain (Fig. 2). The combination of the DEC-205 CP-specific reverse primer 085 with DEC-205-specific forward primers, nested to various parts of DEC-205 ectodomain, yielded major PCR products of the sizes predicted in accordance with the primer combinations used. We also detected slightly smaller (by ~200 bp) minor PCR products, which were most apparent in the primer combinations of 078/085 and 088/085. When the DCL-1-specific reverse primer 086 was used in combination with the same DEC-205-specific forward primers, we detected doublet bands (~200 bp apart), the larger band of which was the predicted size. Sequence analysis indicated that the smaller RT-PCR fragments from DEC-205 itself or the DEC-205/DCL-1 fusion mRNA were amplified from alternatively spliced RNA, lacking
DEC-205 exon 34 (168 bp, described below). Thus, L428 cells express at least two variants of the DEC-205/DCL-1 fusion mRNAs, one with DEC-205 exon 34 fused to DCL-1 exon 2 (a variant termed V34-2) and one with DEC-205 exon 33 fused to DCL-1 exon 2 (a variant termed V33-2) (Fig. 2). Sequence analysis of the fusion junction of V33-2 showed that the junction is in-frame, indicating that V33-2 DEC-205/DCL-1 fusion mRNA is also likely to be translated. The V34-2 encodes the entire DEC-205 ectodomain fused to DCL-1 CRD, TM, and CP. The V33-2 lacks approximately one-third of the C-terminal portion of DEC-205 CRD 10, and the rest of DEC-205 ectodomain is fused to DCL-1.

The DEC-205/DCL-1 Fusion mRNA Is Predominantly Expressed by HRS Cell Lines—To assess DEC-205/DCL-1 fusion mRNA expression, we performed Northern blot analysis in several hematopoietic cell lines (Fig. 3). The DCL-1-specific probe nested within the DCL-1 ectodomain detected a single 28 S ribosomal RNA. The doublets obtained with several sets of primer combinations correspond to alternatively spliced DEC-205 mRNA (see text). SP, signal peptide; CR, cysteine-rich domain; FN, fibronectin type II domain; CRDs, carbohydrate recognition domain; TM, transmembrane domain; CP, cytoplasmic domain.

Diclofalic acid (DCL-1) is a human gene that was previously located to chromosome 2 (22) and more recently mapped to the identical chromosomal band in the NCBI UniGene data base. Using the NCBI Genome BLAST, we identified the human genomic contig NT 005151 containing both DEC-205 and the DCL-1 gene. Our sequence analysis showed that DEC-205 and DCL-1 genes consist of 35 and 6 exons, respectively, and the DEC-205 gene is localized −5.4 kb upstream of the DCL-1 gene (Fig. 4).

The DCL-1 Gene Is Independently Expressed from the DEC-205 Gene—It is possible that the proposed DCL-1 gene is a part of DEC-205 gene and that the DCL-1 mRNA is generated by alternative splicing of DEC-205 mRNA driven by DEC-205 promoter. If this were the case, the DCL-1 5’-untranslated region should contain at least some DEC-205 gene sequences. To assess this possibility, we performed RNA ligase-mediated 5’-RACE using HL-60 total RNA and determined the DCL-1 transcription initiation site (Fig. 3). This procedure is designed to amplify cDNA only from full-length, capped mRNA, and suitable to determine the transcription initiation site. Two rounds of DCL-1-specific PCR amplification of the DCL-1 cDNA yielded a ~250-bp single band regardless of primers (random decamers or DCL-1-specific primer 061) for reverse transcription (Fig. 5A). Sequencing of the 5’-RACE product indicated that DCL-1 transcription initiation site is mapped to 44 bp upstream of DCL-1 translation start codon (ATG, A at +1) located within the 5.4-kb intervening sequence between DEC-205 and DCL-1 gene. Thus, the DCL-1 gene is transcribed independently from DEC-205 gene.

Therefore, the DEC-205 and DCL-1 fusion mRNA variants appear to be generated by cotranscription of both DEC-205 and DCL-1.
DCL-1 genes followed by intergenic splicing to remove the DEC-205 exon 35 alone or exon 34–35 along with DCL-1 exon 2 (V34-2), or DEC-205 exon 33 fused to DCL-1 exon 2 (V33-2) (see Fig. 1). The DNA sequences of DEC-205/DCL-1 fusion mRNA variants and DCL-1 mRNA were submitted to the GenBank™ and assigned the accession number AY184222 (for V34-2), AY314006 (for V33-2), and AY314007 (for DCL-1), respectively. DEC-205/DCL-1 Fusion mRNA Is Translated to the Fusion Protein—We sought to establish whether the DEC-205/DCL-1 fusion mRNA is translated into a fusion protein. We prepared cell lysates from three HRS cell lines (DEC-205 mRNA that is capable of detecting DEC-205/DCL-1 fusion protein bands in HRS cell lines by immunoprecipitation with DEC-205 mAbs and protein G-conjugated beads and Western blot analysis with DCL-1 CP antiserum (data not shown).

To determine the relative abundance of the DEC-205/DCL-1 fusion protein to DEC-205, we developed a sandwich ELISA using the DEC-205 mAbs for capturing and the CP antisera for detection (Fig. 6B). The HRS cell lines express most DEC-205 protein (KM-H2 > L428 > HDLM-2), followed by HEL cells. We detected relatively small amounts of the DEC-205/DCL-1 fusion protein in L428 and HDLM-2 cells, ~30–50 times less than the amount of DEC-205. No fusion protein was detected in the KM-H2 cells, probably because the amount of KM-H2 derived fusion protein is below the detection limit. The negative control, Jurkat, did not show any signal. The relative abundance of both DEC-205 and DEC-205/DCL-1 fusion protein by the ELISA correlated with the immunoprecipitation/Western blot data (Fig. 6A).

**DISCUSSION**

Cotranscription and intergenic splicing is a rare event in mammalian cells, and there are only a small number of reports describing the presence of fusion mRNA. These include MDS/
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...carry out the fusion of DEC-205 and DCL-1, respectively. The signals were detected with anti-DEC-205 CP (for DEC-205) or anti-DCL-1 CP (for DCL-1), respectively. The signals were detected with O-phenylenediamine dihydrocholate at 492 nm.

EVI1 (29), galactose-1-phosphate uridyltransferase, and interleukin-11 receptor genes (30), Prnd/Prnp (31) and P2Y1/SSF1 (32). None of these reports, however, examined whether the fusion mRNA is translated endogenously into cognate fusion protein. Here we describe another cotranscription and intergenic splicing of two juxtaposed genes, encoding two type I transmembrane C-type lectin receptors DEC-205 and DCL-1, respectively. Furthermore, we demonstrated for the first time that DEC-205 and DCL-1 fusion mRNA is translated endogenously into DEC-205/DCL-1 fusion protein. Here we describe another cotranscription and intergenic splicing of two juxtaposed genes, encoding two type I transmembrane C-type lectin receptors DEC-205 and DCL-1, respectively. Furthermore, we demonstrated for the first time that DEC-205 and DCL-1 fusion mRNA is translated endogenously into DEC-205/DCL-1 fusion protein. DEC-205 is a putative antigen uptake receptor expressed on dendritic cells (17, 33) and potent APCs, which initiate and direct immune responses (reviewed in Refs. 34–37). DEC-205 belongs to the macrophage mannose receptor family of endocytic receptors that include the prototype macrophage mannose receptor (MMR) (38, 39), phospholipase A2 receptor (PLA2R) (40, 41) and Endo180 (42, 43). The ectodomain of these receptors contains several domain structures, including a cysteine-rich domain, fibronectin type II domain, and multiple CRDs (10 for DEC-205 and 8 for others). The MMR, PLA2R, and Endo180 exhibit C-type lectin activity (39, 40, 42), however, the DEC-205 ligands have yet to be identified. The cytoplasmic domain of these receptors contains either Tyr-based (in MMR, PLA2R, and DEC-205) (39, 44, 45), di-aromatic amino acid-based (46), or di-hydrophobic amino acid-based (in Endo180) (47) motifs to facilitate their endocytosis to transport cognate ligand intra-cellarly. Although there are potential Ser and Thr phosphorylation sites within the CP of these lectins, no phosphorylation of these sites has been reported. In addition, the DEC-205 CP contains a cluster of acidic amino acids (EDE) that targets late endosomes, where loading of proteolytically processed antigenic peptides to major histocompatibility complex class II occurs (45). DCL-1 is a unique type I transmembrane C-type lectin in that DCL-1 ectodomain contains only one CRD, whereas other type I transmembrane C-type lectins contain more than one domain (e.g. selectins and MMR). DCL-1 CP contains several putative motifs, including a Tyr-based internalization, a cluster of acidic amino acids, and Ser and Tyr phosphorylation motifs, suggesting that DCL-1 CP mediates not only endocytosis and late endosome targeting but also signaling.

The genes encoding DEC-205 (LY7) (17) and DCL-1 (KIAA0022) (22) are juxtaposed within chromosome band 2q24 and are separated by only ~5.4 kb (Fig. 4). These are independent genes, because DEC-205 and DCL-1 mRNA are each expressed independently in hematopoietic cell lines (Fig. 3). The 5’-RACE experiment mapped the DCL-1 transcription initiation site at 44 bp upstream of DCL-1 translation start codon (Fig. 5). Furthermore, our recent luciferase reporter assay studies showed that both 5’-proximal promoters of DEC-205 and DCL-1 have independent promoter activity (data not shown). The DEC-205 promoter may drive the cotranscription of the DEC-205 and DCL-1 genes to produce the 9.5-kb DEC-205/DCL-1 fusion mRNA. This would result from leaky termination of DEC-205 transcription, a mechanism suggested to explain the cotranscription of galactose-1-phosphate uridyltransferase and interleukin-11 receptor genes (30). However, this seems unlikely, because the expression of DEC-205 mRNA did not correlate to that of DEC-205/DCL-1 fusion mRNA. Interestingly, all HRS cell lines tested expressed the 9.5-kb DEC-205/DCL-1 fusion mRNA, but not 4.2-kb DCL-1 mRNA, whereas other myeloid and B cell lines expressed 7.5-kb DEC-205 and/or 4.2-kb DCL-1 mRNA (Fig. 3), suggesting that expression of DEC-205/DCL-1 fusion mRNA is highly regulated. It is intriguing to speculate that HRS cell lines express certain transcription factors that may control cotranscription of DEC-205 and DCL-1 genes.

At mRNA levels, we identified two DEC-205/DCL-1 fusion mRNA variants (V34-2 and V33-2) different by the presence of the DEC-205 exon 34. The deletion of exon 34 appears to be the only alternative splicing that occurs naturally in DEC-205 gene transcription (Fig. 2). The fusion junctions in the V34-2 and V33-2 DEC-205/DCL-1 fusion mRNA are in-frame, suggesting both transcripts are translated.

What would be the functional difference between DEC-205 and DEC-205/DCL-1 fusion protein? Because the fusion protein contains DCL-1 CP, not DEC-205 CP, it is conceivable that DEC-205 ligand (currently unknown) to DEC-205/DCL-1 fusion protein would induce distinct signals from that binding to DEC-205. Further study of DEC-205 and DCL-1 is required to elucidate the function of these two C-type lectin receptors as well as DEC-205/DCL-1 fusion protein.

In this study, we used three independent HRS cell lines (L428, HDLM-2, and KM-H2) and showed that DEC-205/DCL-1 fusion mRNA is predominantly expressed in these HRS cell lines (Fig. 3) and that the mRNA is translated into a DEC-205/DCL-1 fusion protein (Fig. 5). We are currently investigating the presence of DEC-205/DCL-1 fusion mRNA in HL-affected lymph nodes in situ. If the expression of DEC-205/DCL-1 fusion protein is confirmed in vivo in HL, then the fusion protein may become relevant as a new target for antibody or T cell mediated immunotherapy for HL.
