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Sustained expression of a neuron-specific isoform of the *Taf1* gene in development stages and aging in mice

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ABSTRACT

TATA-box binding protein associated factor 1 (TAF1) protein is the largest and the essential component of the TFIID complex in the pathway of RNA polymerase II–mediated gene transcription, and it regulates transcription of a large number of genes related to cell division. The neuron-specific isoform of the *TAF1* gene (*N-TAF1*), which we reported previously, may have an essential role in neurons through transcriptional regulation of many neuron-specific genes. In the present study, we cloned the full-length cDNA that encodes the mouse homologue of N-TAF1 (N-Taf1) protein. By carrying out of real time RT-PCR, we investigated the expression analysis of the *N-Taf1* mRNA in mouse tissues and cell lines. As well as the human *N-TAF1*, the *N-Taf1* showed limited expression in the brain and neuroblastoma, whereas *Taf1* expressed elsewhere. Furthermore, in mouse embryo head or mouse brain, mRNA expression of *TAF1* changes dramatically during development but *N-Taf1* showed sustained expression. Our result suggests that the *N-Taf1* gene has an important role in non-dividing neuronal cell rather than in cell division and proliferation during neurogenesis.

1. Introduction

TATA-box binding protein associated factor 1 (TAF1) is the largest subunit of transcription factor IID (TFIID), which is composed of TATA-box binding protein (TBP) and 13 TATA-box binding protein associated factors (TAFs) [1,2]. Binding of TFIID to the core promoter elements is required for assembly of a functional transcription initiation complex. TFIID also serves as a co-activator by directly transmitting signals from sequence-specific activators to other components of the basal transcription machinery [3–5]. Moreover, TAFs directly activate selected genes *in vivo* [6–8]. TAF1 appears to function as a major scaffold by which TBP and other TAFs interact in the assembly of TFIID. TAF1 plays a critical role in the regulation of cell growth [6]. Also TAF1 possesses intrinsic protein kinase activity [9], histone acetyltransferase activity [10] and ubiquitin-activating and conjugating activity [11]. And mutational analysis demonstrated that kinase activity of TAF1 is important for the progression through the G1 phase [12].

We previously found that *TAF1* gene is the disease causative gene of X-linked recessive dystonia-parkinsonism showing severe neurodegeneration in striatum (XDP/DYT3; MIM314250) [13]. In the brain of patients with XDP/DYT3, the expression of a neuron-specific isoform of the *TAF1* is significantly decreased. Therefore, the neuron-specific isoform of the *TAF1* gene, named *N-TAF1*, may have an essential role in neuronal survival in the striatum through transcriptional regulation of many neuron-specific genes. Although, we found the expression of *N-TAF1* mRNA has been characterized in human brain tissues and neuroblastoma [13], a little information is available about its functions in the non-dividing neuronal cells. In the present study, we cloned the full length ORF of mouse homologue of *N-TAF1* (*N-Taf1*), and investigated if *N-Taf1* expression has tissue-specificity in mouse various tissues and cell lines using probe-based real time RT-PCR. In addition, we examined the altering gene expression levels of *Taf1* and *N-Taf1* with development stages and aging in the mice embryo head or brain.

2. Materials and methods

2.1. Animals

Three adult male BALB/c mice were used for expression analysis in tissue and adult or pregnant ICR mice were used for developmental analysis. This study was performed in accordance with the PHS Policy on Humane Care and Use of Laboratory Animals, the NIH Guide for the Care and Use of Laboratory Animals, and the Animal Welfare Act (7 U.S.C. et seq.). All experimental

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protocols were approved by the Institutional Animal Care and Use Committee of the University of Tokushima.

2.2. Long RT-PCR analysis

Total RNA was isolated from adult BALB/c mice brain using RNeasy Lipid Tissue Midi Kit (QIAGEN, CA, USA). And then, mRNA was purified with Dynabeads mRNA purification Kit (Dynal Biotech LLC, WI, and USA) according to the manufacturer's protocol. For the first strand synthesis, mRNA was reverse transcribed with the gene specific mTA2_r_8003 primer (5′-ACTTACTTGGCAAGCAGAGTCC-3′) and SuperScript III Reverse Transcriptase (Life Technologies Corp., CA, and USA). And then, fragment PCR were performed by use of the long RT products as a template. The PCR mixture contained 2 μl of the first-strand cDNA, 0.2 mM of each dNTP, 1 mM of each primer, 1× GeneAmp PCR buffer, and 2.5 Units of AmpliTaq Gold DNA polymerase, in a 50 μl total volume. The PCR conditions were 9 min at 95 °C, followed by 40 cycles at 95 °C for 45 s, 58 °C for 45 s, and 72 °C for 60 s. Details of the primers used in this fragment amplification are shown in Table 1. Each PCR products were analyzed by agarose gel electrophoresis and direct sequencing. And the mTA14 fragment with fluorescent-labeled forward primer (TA2_f_4789) and non-labeled reverse primer (TA2_r_5178), expecting to include human *N-TAF1* specific exon, was verified by capillary electrophoresis using the ABI PRISM 310 (Life Technologies Corp., CA, and USA).

2.3. Cloning of mouse N-TAF1 and mouse TAF1 ORF

For cloning of ORF encoding mouse *N-TAF1* (*N-Taf1*) and mouse *TAF1* (*Taf1*), heminested PCR was performed using mouse brain CapSite cDNA dT library (NIPPON GENE CO., LTD, Tokyo, Japan) as a template. The CapSite cDNA libraries consist of cDNAs in which the 5′ cap structure (m7Gppp) of eukaryotic mRNA is replaced with a synthetic oligoribonucleotide to label the 5′ end of the cDNA, enabling identification of the 5′ end sequence by PCR [14]. Long PCR was performed with Advantage 2 Polymerase Mix (Clontech Laboratories, Inc., CA, and USA). We used a primer set for the first PCR—mTA2_f_10 (5′-AGGGAGCGCTGCAAGTC-3′) and mTA2_r_5948 (5′-TGAGGAAGAGTGAGTCTGCTCTC-3′)—and then used a set for the second heminested PCR—mTA3_f_38 (5′-ACTGACGTATTTTTTCTTTCCCG-3′) and mTA2_r_5948. The second PCR product was separated on agarose gel and recovered by using QIAEX II Gel Extraction Kit (QIAGEN, CA, USA) followed by direct sequencing with 16 internal primers. And then, the purified PCR product was cloned into pGEM-T easy vector (Promega Corp., WI, and USA). The insert was confirmed by sequencing with 16 internal primers same as above.

2.4. Quantitative RT-PCR analysis

Total RNA was prepared from seven tissues (brain, heart, spleen, lung, liver, thymus and stomach) of adult BALB/c mice and three cell lines (Neuroblastoma Neuro-2a, Cerebellum astrocyte C8-D1A and Fibroblast L). In addition, we isolated total RNA from ICR mouse embryo head (10.5 dpc) and brain (17.5 dpc, 1 day, 1 week, 3 weeks, 5 weeks, 7 weeks, 15 weeks, 40 weeks). According to the manufacturer's instructions, we used RNeasy Kit, RNeasy Lipid Tissue Kit or RNeasy Fibrous Tissue Kit (QIAGEN, CA, USA), respectively. The RNA was treated with RNase-Free DNase I (QIAGEN, CA, USA) to remove the residual genomic DNA, and then reverse transcribed by use of random hexamers with a TaqMan Reverse Transcription Reagents kit (Life Technologies Corp., CA, and USA). The reverse transcription mixture was amplified using an ABI PRISM 7000 Sequence Detection System in accordance with the standard procedure. Sequences of the primers and probes for *N-Taf1* were 5′-GAATTAGAAAGTCTGGACCCAATGAC-3′ (sense), 5′-GCTCTCATCTTGATATACAGAAGCATCT-3′ (antisense) and 5′-FAM-CTCAGGCTAAGCCTC-MGB-3′ (probe). For *Taf1*, we used the same primer set and the *Taf1* specific probe (5′-FAM-ACACCTCAGCCTCC-MGB-3′), which is designed to detect all isoform sequences not containing *N-Taf1* specific exon. The final concentrations of probes and primers are 250 and 900 nM, respectively. We also purchased control TaqMan probes for 18S rRNA (4319413E) from Life Technologies. Quantity was calculated every time by use of a standard curve for each well.

Table 1
Primer sequences of oligonucleotides used for PCR.

Fragment Name	Forward primer		Reverse primer		Amplicon size (bp)
	Name	Sequence	Name	Sequence	
mTA01	mTA2_f_10	AGGGAGCGCTGCAAGTC	mTA2_r_411	CACCTCGTTGATGTCAGAATAGTC	402
mTA02	mTA2_f_381	TGCTGTGGACTATTCTGACATC	mTA2_r_783	GGCATCATGCTGCATAATC	403
mTA03	mTA2_f_762	TGGGATTATGCAGCATGATG	mTA2_r_1191	AACACCTAGCATATCATACCACAGC	430
mTA04	mTA2_f_1121	TGGATACCAAGCCAAGAGTG	mTA2_r_1586	TCCCAAATGATATTGTCCTCC	466
mTA05	mTA2_f_1532	CCATTGATAATGAAGATCTGGTATATG	mTA2_r_1923	CCGTAATTCTACAGCTGGAATTG	392
mTA06	mTA2_f_1877	TTGGAGGAAATATTATCCAGCAC	mTA2_r_2318	AGGAAAGGCGATGTATGGC	442
mTA07	mTA2_f_2264	GAGCCCCAGATTGCAAATAC	mTA2_r_2624	GCTTTCTTTATATCTTCCATCCG	361
mTA08	mTA2_f_2576	AGAGTAAAGATAGGCCACGGAG	mTA2_r_3004	CACAGCCTGTAGGATCTGCTAC	429
mTA09	mTA2_f_2958	CAAATGTCTCCTGGAGGTGAC	mTA2_r_3362	GATGACAAAACCTTGTTCTGTAGG	405
mTA10	mTA2_f_3308	GTTACAAAGAGGAATGTCAGCG	mTA2_r_3725	GTGGTCCGTATGCGTACATAAG	418
mTA11	mTA2_f_3657	GGAATATGTTCGTTGTGAGACAG	mTA2_r_4130	TCATCTGCACTCTCAATTAGCTG	474
mTA12	mTA2_f_4075	AAGGTTGAAGGGACCAAGATC	mTA2_r_4479	TAGCTCCAAATGTTCTCTGAATTC	405
mTA13	mTA2_f_4397	CAATGGATCTACAGACACTCCG	mTA2_r_4848	CAGGTTTACGTCGTCTAGAAAGC	452
mTA14	mTA2_f_4789	AATATCTCCAAGCACAAATACCAG	mTA2_r_5178	CTGCTTTTCTGAAGTAGCACTG	387
mTA15	mTA2_f_5126	ATGAGAGCAATCTGTCTGTCTTG	mTA2_r_5541	CTCCTCATAGCTCCCATAACTG	416
mTA16	mTA2_f_5495	CCCGTGGTTTGGAGGATAG	mTA2_r_5948	TGAGGAAGAGTGAGTCTGCTCTC	454
mTA17	mTA2_f_5893	GCAAGTTCTTTTCCCTAACCACTAC	mTA2_r_6281	GCAGTCTTTGCCCTAAGGTAC	389
mTA18	mTA2_f_6200	ATAGGTGGTCATGTAAATGTCCTC	mTA2_r_6629	TGCTGAGATGGTACTCTATAACTTCTC	430
mTA19	mTA2_f_6575	TCTAGAGGCTACCTGAATGCTG	mTA2_r_7016	GACTTCTAGACAAACTGAATCATACATG	442
mTA20	mTA2_f_6952	ATGTTGAATGAAGATAGAAATTGAGTC	mTA2_r_7392	CAAGTGCCAACTGGTTATGAAG	441
mTA21	mTA2_f_7342	ATCTTCAGAAAGGGGTGTGATC	mTA2_r_7658	TGAACTCAGAGATGTGCCTTG	317
mTA22	mTA2_f_7610	GCAGTTTAAGACAGCTGGGTG	mTA2_r_8003	ACTTACTTGGCAAGCAGAGTCC	394
mTA01	mTA2_f_10	AGGGAGCGCTGCAAGTC	mTA2_r_411	CACCTCGTTGATGTCAGAATAGTC	402
mTA02	mTA2_f_381	TGCTGTGGACTATTCTGACATC	mTA2_r_783	GGCATCATGCTGCATAATC	403

3. Results

3.1. Long RT-PCR and the alternative exons of mouse Taf1

The long RT-PCR experiment using primer sets for the entire coding region of mouse *Taf1* is shown in Fig. 1A. The cDNA fragment of *Taf1* with the long RT primer on the end of exon 38 was synthesized. Fragment PCRs were performed using long cDNA to search the *Taf1* variations and we found that four fragments had multiple bands (mTA02, mTA14, mTA21, and mTA22 were indicated by red bars) and other fragments appeared as single bands (indicated by green bars). There are some isoforms reported around mTA02 region, which were expressed in general tissue. The mTA21 and mTA22 regions are located near three prime untranslated region (3′UTR), which we needed to stay focused on only mTA14 region. The mTA14 fragment labeled with fluorescent showed two peaks differed with 6 nucleotides by fragment analysis (Fig. 1B). Nucleotide sequence of shorter PCR product was identical to reported *Taf1* cDNA. But the longer PCR product showed same nucleotide sequence as human *N-TAF1* specific exon sequence (GenBank ID: AB300418) (Fig. 1C). This reveals that the longer PCR product is neuron specific mouse *N-TAF1* (*N-Taf1*).

3.2. Full-length cloning of Taf-1 and N-Taf1 ORF

The cloning of *Taf1* ORF and *N-Taf1* ORF was performed (Fig. 2A) to determine the complete structure of the isoform containing the 6 bases insertion. A single band was detected (Fig. 2B) by the heminested PCR, and the PCR product was analyzed by direct-sequencing method with use of 16 redundant internal sequencing primers. Around mTA14 region, we found incorrectly called, mixed base in electropherogram (Fig. 2C). The position of this mixed base was consistent with the 6 bases insertion. Then, we cloned PCR product into TA cloning vector and analyzed twenty-six clones by sequencing. As a result of sequencing, 14 clones (53.8%) had the 6 bases insertion in mTA14 region (Fig. 2D). The complete coding region of *N-Taf1* was deposited in GenBank (GenBank ID: AB299229). The complete coding sequence of *N-Taf1* gene is translated into 1,893 amino acids with an insertion of two amino acid residues, alanine (A) and lysine (K), which is identical to that of human *N-TAF*1 (GenBank ID: AB300418).

3.3. Neuron-specific expression of N-Taf1 and its expression pattern in development stages and aging

We examined the mRNA expression levels of *Taf1* and *N-Taf1* in the various tissues (brain, heart, spleen, lung, liver, thymus and stomach) and mouse cell lines (Neuroblastoma Neuro-2a, Cerebellum astrocyte C8-D1A and Fibroblast L), using quantitative RT-PCR by designing specific probes. *Taf1* mRNA was expressed in most tissues and cell lines, but expression of *N-Taf1* was highest in the brain and Neuroblastoma N2a cell lines. This limited expression in the brain and neuron showed *N-Taf1* could be neuron specific gene.

Using same probe we aimed to study the expression of these genes in the mouse brain in accordance with developmental stages. We analyzed the expression pattern of *Taf1* and *N-Taf1* in mouse embryo head (10.5 dpc) and brains (17.5 dpc and 1 day to 1, 3, 5, 7, 15, 40 weeks). The *Taf1*, expressed in the most tissues, showed highest expression in the embryonic stages and gradually decreased until 3rd week. Then, from 3 to 40 weeks, the expression level of *Taf1* has no change. In contrast, almost no expression of *N-Taf1* was found at 10.5 dpc in the embryo head, but there is a good amount at 17.5 dpc in the brain. The expression of *N-Taf1* was sustained from 17.5 dpc to 40 weeks in the brain (Fig. 4).

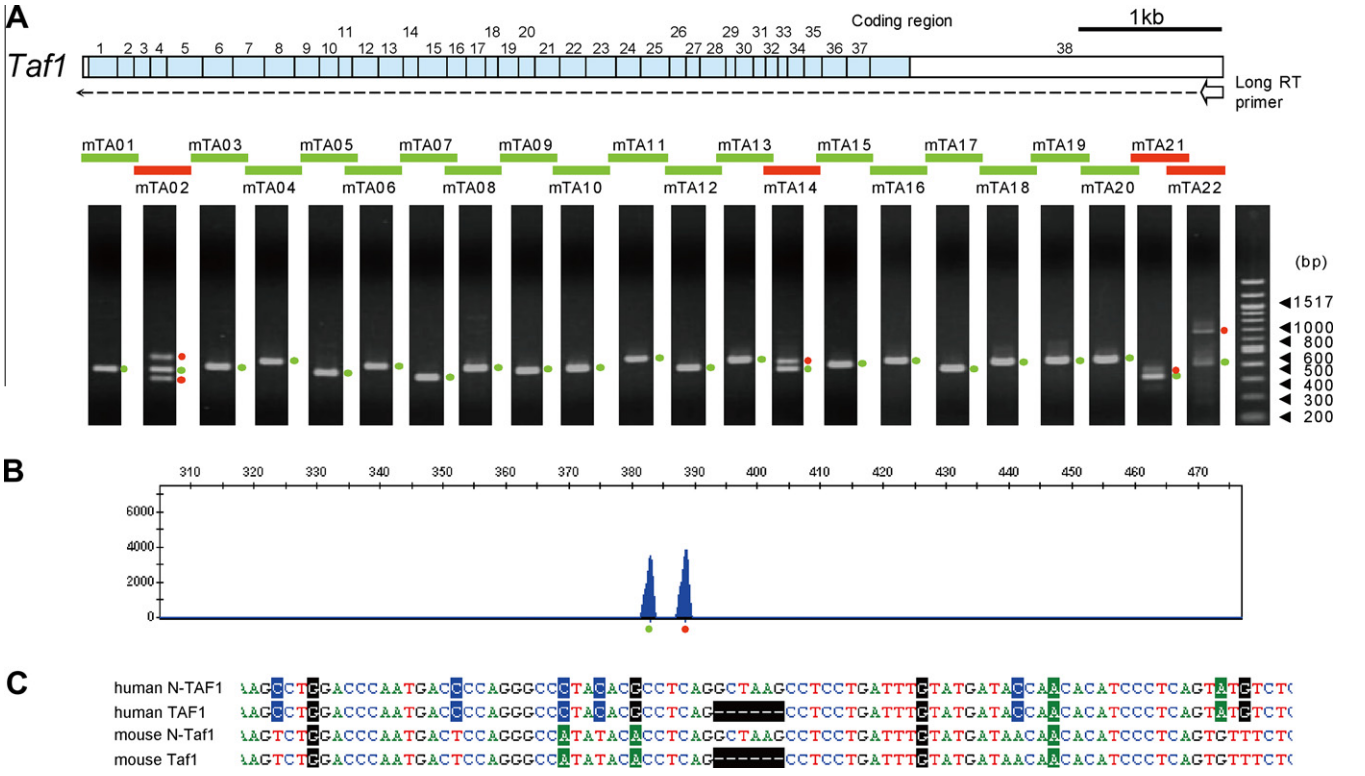

Fig. 1. Long RT-PCR and the alternative exons of *Taf1*. (A) Long RT-PCR analysis. The broken line indicates an expected cDNA fragment of *Taf1* with the long RT primer on the end of exon 38. (B) Fragment analysis. The fluorescently labeled mTA14 fragments showed two peaks with 6 bases difference by capillary electrophoresis. (C) The nucleotide sequence of mTA14 fragment. The longer PCR product had 6 nucleotides sequence same as human *N-TAF1* specific exon.

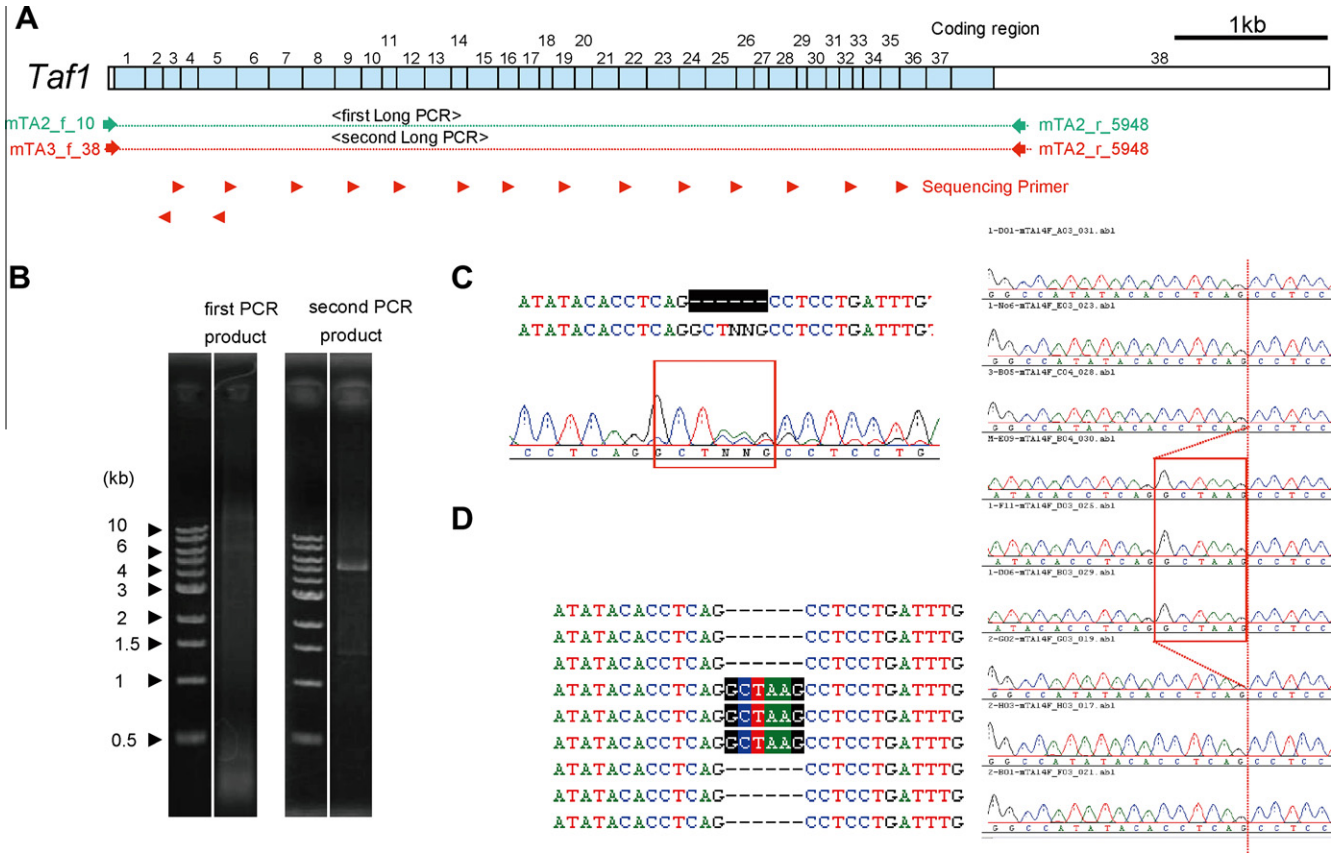

Fig. 2. Cloning of *Taf1* and *N-Taf1* ORF. (A) The cDNA cloning of mouse *TAF1* (*Taf1*) and *N-TAF1*(*N-Taf1*) sequence. The long PCR product was obtained from mouse brain CapSite cDNA dT by heminested PCR. The complete DNA sequences of these long products were determined by a PCR direct-sequencing method by use of 16 redundant internal sequencing primers (red triangles). (B) From the second PCR, products of sufficient quantity and quality for PCR product direct-sequencing were amplified. (C) The electropherogram of PCR product direct-sequencing with the second PCR product. Around mTA14 region, incorrectly called mixed base was found. (D) *N-Taf1* specific 6 bases insertion in cloned plasmid. The clones in the fourth, fifth and sixth from the top of (D) have *N-Taf1* specific 6 bases insertion. (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article.)

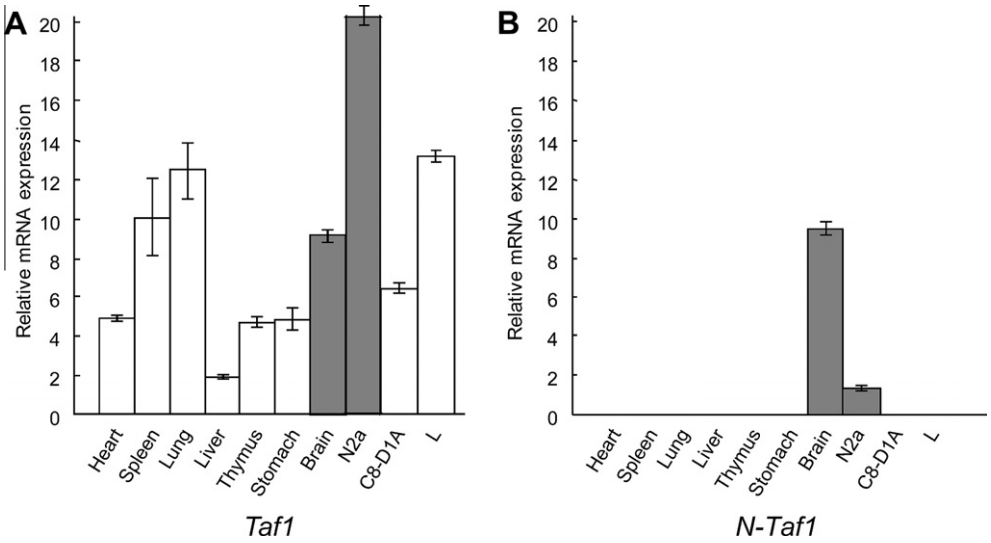

Fig. 3. Expression of the *Taf1* and *N-Taf1* in various tissues and cell lines of mouse. Expressions of *Taf1* (A) and *N-Taf1* (B) are shown relative to the expression of 18S rRNA (as an internal control). The label "relative mRNA expression" means relative mRNA expression level to 1/20 × 18S rRNA. Values are expressed as means ± SD ($n = 3$).

4. Discussion

In the present study, we have shown that the gene expression of mouse *N-Taf1* gene, include additional two amino acids as well as human *N-TAF1* gene, is limited in the brain. This is consistent with our previous findings [13]. These results indicate that *TAF1* has a neuron-specific isoform and specific exon sequence of that isoform have revealed highly conservation between species. Sako et al. showed that in the rat brain, N-TAF1 protein appears as a nuclear protein within the neurons [15]. Our result was consistent with Sako's findings. Furthermore, finding from fragment analysis of mTA14 region (Fig. 1B), count of cloned *N-Taf1* (Fig. 2D) and quan-

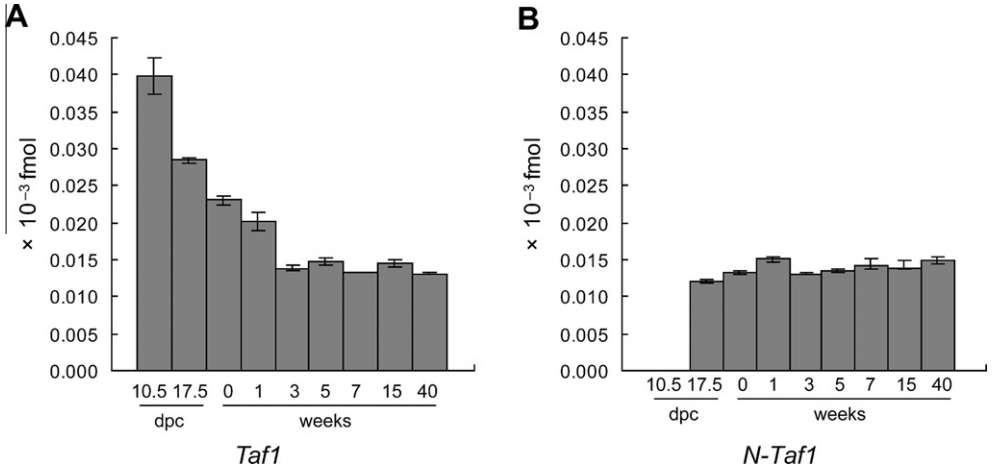

Fig. 4. Expression of the *Taf1* and *N-Taf1* with Mouse Development and aging. The abundances for *Taf1* (A) and *N-Taf1* (B) were determined using quantitative RT-PCR by use of each clone of known concentration in the linearized plasmid as a standard. Values are expressed as means ± SD (n = 3).

titative RT-PCR analysis (Figs. 3 and 4) indicate that expression revel of *N-Taf1* mRNA is higher in the brain rather than neuroblastoma, suggesting that *N-Taf1* could be functional in the differentiated neurons.

The TAF1 protein is the largest and the essential component of the TFIID complex in the pathway of RNA polymerase II–mediated gene transcription, and it regulates transcription of a large number of genes related to cell division and proliferation. Our result, the *Taf1* expressed highly in the early embryogenesis of mouse embryo head and brain, would be reasonable because of these previous findings. In contrast, *N-Taf1* showed the different expression pattern as *Taf1* (Fig. 4). *N-Taf1* showed almost no expression at early embryonic stage but good expression at 17.5 dpc, which may explain that *N-Taf1* expressed in well-defined CNS and this neuron-specific isoform is responsible for some genes express between 10.5 and 17.5 dpc. Then, the expression level of *N-Taf1* is sustained even in aged brain. TAF1 pre-mRNA generates a number of TAF1 isoforms. Metcalf and Wassarman are using Drosophila as a model system to study the biochemical and biological roles of TAF1 protein isoforms [16]. These studies have shown that TAF1 isoforms have different DNA binding specificities and are differentially expressed during Drosophila development [16–18]. *N-TAF1* may also bind with own target DNA sequence. The role of the *N-TAF1* in non-dividing neuronal cells is still unclear. But the differences in the expression pattern between *Taf1* and *N-Taf1* suggest that *N-Taf1* may have an important role, such as protectoral function in differentiated neurons, rather than in cell division and proliferation during neurogenesis.

In conclusion, we identified the mouse homologue of *N-TAF1* (*N-Taf1*) and demonstrated the expression pattern of N-Taf1 mRNA in mouse embryo head and brain. The different expression pattern between Taf1 and N-Taf1 mRNA indicated that N-Taf1 might play an important role in non-dividing neuronal cells.

Acknowledgments

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