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Running title: Periostin splice variants in cancer

A splice odyssey: periostin's journey through the sea of crabs

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Periostin is an extracellular matrix protein involved in processes ranging from early embryonic development to life-threatening cancer progression. Instead of acting as a single entity, it exists in numerous alternatively spliced isoforms, primarily differing in their C-terminal region, which drives diversification and context-dependence in its biological functions. Despite growing interest, inconsistencies in nomenclature and fragmented data still complicate the interpretation of isoform-specific roles. Therefore, the aim of this review is to provide a structured overview of periostin isoforms, focusing on their roles in development and carcinogenesis, while addressing challenges in their nomenclature. During embryogenesis, expression of periostin is dynamically regulated across developmental stages and tissues. Distinct isoforms exhibit specific patterns, both spatially and temporally, that reflect specialized functions during organogenesis (particularly in the cardiac, neural, and skeletal systems), and as experimental evidence points out, certain exons play critical roles in morphogenesis. Regulatory mechanisms influencing these patterns persist beyond development and are often changed in pathological conditions. In oncological diseases, expression of periostin isoforms varies widely between tumor types and microenvironments. Many isoforms are present in malignant as well as in adjacent non-malignant tissues, with differences in their relative abundance. Functionally, exon 17-containing variants tend to influence tumor cell-intrinsic properties, while variants with exon 21 are more closely linked to stromal interactions and modulation of the tumor microenvironment. Interesting is also the fact that periostin can exert both tumor-promoting and tumor-limiting effects, depending on the isoform and biological context. Taken together, periostin isoforms represent a complex regulatory system whose better understanding and standardized classification may enhance their potential in diagnostics and therapy.

Key words: periostin; alternative splicing; exon; development; cancer

Periostin is a matricellular protein that plays a crucial role in tissue remodeling, fibrosis, and, remarkably, cancer progression [1-3]. In recent years, it has become known as an important regulator of the tumor microenvironment, influencing processes such as cell survival, invasion, and metastasis. However, increasing evidence suggests that periostin does not function as a single, uniform protein but rather as a family of alternatively spliced isoforms with potentially different biological roles [4-6]. The existence of multiple periostin isoforms, that primarily vary in their C-terminal region, adds another layer of complexity to their function (Figure 1, Table 1) [7]. These isoforms are not equally expressed across tissues and disease states, including numerous types of cancer, where they may exert divergent or even opposing effects [4, 5, 8]. Inconsistencies in nomenclature and fragmented findings across studies, however, have made it challenging to draw clear conclusions about their specific roles.

Periostin was firstly identified in 1993 as osteoblast-specific factor 2 and its nucleotide sequence was described as consisting of a 5′untranslated region with a length of 18 bp, an open reading frame of 2436 bp, and a 3′untranslated region containing 733 bp. The encoded protein was reported to be 811 amino acids long and composed of an N-terminal sequence, a cysteine-rich region, four domains with homology to fasciclin I, and a C-terminal region. Screening human cDNA libraries using mouse cDNA as a probe identified five human variants of osteoblast-specific factor 2 that differ only in the C-terminal region. The mouse and human proteins are highly conserved, with differences likewise mainly localized to the C-terminal region. This region shows in-frame insertions and deletions, implying it is subject to alternative splicing [9]. In 1999, osteoblast-specific factor 2 was renamed *periostin* based on its apparent tissue specificity. Afterwards, the presence of several periostin isoforms was confirmed also in mice with individual transcripts differing from each other by the presence or absence of one or more of the six sections (corresponding to peptides encoded by one or two exons) all localized within the C-terminal domain. The observation that these isoforms are not uniformly expressed through different cell lines [7], together with the identification of distinct sequences encoding human osteoblast-specific factor 2 in placental and osteosarcoma cDNA libraries [9], marked the beginning of an odyssey in the study of periostin isoforms, which have been since that time extensively investigated for their temporal, spatial, and disease-specific expression. This journey was challenged not only by inconsistencies in nomenclature, which often complicated direct comparison of described isoforms but also by contradictory findings reported in different studies [10-12]. Therefore, the aim of this work is to provide a comprehensive, structured overview of periostin isoforms, with a focus on their role in cancer, while addressing discrepancies in nomenclature and emphasizing their potential as diagnostic and therapeutic targets. In this review, to provide a consistent framework for comparison, periostin isoforms are referred to based on exon composition using a Δ-exon nomenclature (e.g., Δ^{17} indicates the absence of exon 17). This approach facilitates comparison between studies that have used different systems for naming individual periostin isoforms (resulting in several various designations for the same isoform), which are summarized and cross-referenced in Supplementary Table S1.

Periostin and development

In the human uterus, periostin is expressed in the endometrium and exhibits dynamic changes during the menstrual cycle [13, 14]. After fertilization of the ovum, periostin levels in the serum of gravid women correlate with gestational age, proposing a role in implantation and early embryonic development. Higher levels of periostin detected in evolutive pregnancies compared to miscarriage cases further encourages its functional importance in this process [15, 16]. Periostin isoforms exhibit distinct expression patterns in time and space during embryonic development. This was comprehensively documented by Zhu et al., who analysed the distribution of periostin variants differing in their C-terminal composition in mouse embryos. Their work revealed that isoform(s) containing exon 17 (thought to be the periostin-like factor) are broadly expressed during early development, particularly in neural tissues, cartilage, and the heart, with neural expression declining at later stages. In contrast, isoform(s) containing exon 21 are largely absent from neural tissues and become more prominent in mesenchymal structures, especially in developing skeletal tissues.

These observations indicate that individual periostin isoforms may contribute to various embryogenesis-related processes depending on both developmental stage and tissue context [17].

The complexity of periostin isoform expression was highlighted already in earlier studies-in 2004, periostin-like factor was first mentioned as periostin isoform lacking exon 21 [18], demonstrating the presence of multiple variants during cardiac development and osteoblast differentiation. The number and identity of detected isoform(s) vary considerably depending on the experimental approach used in particular study. This variability has been attributed to differences in detection sensitivity, incomplete coverage of alternatively spliced regions in RT-PCR assays, and the lack of antibody recognition for specific isoform epitopes. These limitations underscored the need for precise isoform characterization and standardized terminology in future studies as well [18]. Interpretation of periostin isoform expression is strongly influenced by the methodological approach used. For example, antibody-based detection targeting peptides encoded by specific exons (such as exon 17 or exon 21) does not identify exclusive isoform directly, but rather group of variants containing the respective exon. On the other hand, sequencing-based methods enable precise characterization of individual splice variants, such as those lacking exon 21 (Δ^{21}). These differences stress the importance of carefully considering experimental design when comparing results across studies and further emphasize the need for standardized nomenclature in research focused on periostin isoforms [17, 18] (Supplementary Table S1).

Periostin isoforms are dynamically regulated during embryonic stages, with distinct temporal expression patterns reflecting changing functional requirements. In murine heart, periostin expression can be detected as early as embryonic day 8.5, with a clear shift in isoform composition over time - while shorter isoforms predominate during early embryogenesis, longer variants become more abundant during later prenatal stages, followed by a postnatal shift back toward shorter forms. Overall periostin expression increases during development, peaks in the neonatal period, and subsequently declines in adulthood [19]. Similar pattern of dynamic regulation has been detected in craniofacial tissues - the highest periostin levels were found in newborns while in adult tissues there was a significant decrease. Moreover, this shift was accompanied by an isoform switch from predominantly long variants in embryonic stages to shorter forms in adulthood. This transition has been consistently observed across multiple craniofacial structures, including teeth and periodontal tissues, suggesting that periostin isoform composition is tightly linked to tissue maturation and remodeling processes [20].

Genetic models further favor the functional importance of periostin isoforms during development. Knock-out studies targeting specific exons (e.g., exon 17 and exon 21) revealed significant impairments in craniofacial and skeletal growth, accompanied by reduction in body weight. Notably, based on these studies, exon 17 appears to play a particularly critical role by regulating key osteogenic and chondrogenic pathways [21]. In addition to exon-specific models, studies using periostin-null mice further draw attention to its biological importance. Despite the fact that these animals appear more-or-less normal at birth, postnatally they develop pronounced abnormalities, including growth retardation, skeletal defects, increased mortality, and severe periodontal and enamel abnormalities. The delayed onset of these phenotypes indicates that periostin may be required for developmental processes established *in utero* but manifested after birth. Alternatively, its

function during embryogenesis may be partially compensated by other gene(s) with overlapping or redundant roles [22].

Despite majority of the research on periostin during development so far has been conducted in mouse models, studies in human tissues manifest a similarly complex and context-dependent pattern of isoform expression. In contrast to the predominance of hard-tissue studies in mice, human studies have focused more on visceral organs, such as lung and kidney. Analyses of human kidney tissues have identified several periostin isoforms with dissimilar expression profiles across developmental and pathological states. Initial studies detected six isoforms, including the full-length variant, with differing abundances in fetal, adult, and neoplastic samples, suggesting context-specific roles in embryogenesis, normal physiology, and tumorigenesis [23]. Following analyses enriched this observation by identifying up to eight isoforms, some of which were exclusively present in fetal kidney tissue (full-length; $\Delta^{17,18}$, Δ^{21}, Δ^{18}, and $\Delta^{17,18,19}$) and absent in both adult and cancer samples. These development-specific variants are likely associated with dynamic extracellular matrix remodeling processes characteristic for organogenesis [8]. A comparable, yet not identical, pattern has been recognized in the developing lung - similar number of periostin isoforms has been detected, but their composition differed tissue-specifically. Certain variants present in the fetal kidney (Δ^{18}) are absent in the lung, and *vice versa* (isoform $\Delta^{18,21}$), with some isoforms uniquely detected in fetal lung tissue but not in adult or cancerous samples [24]. Taken together, these findings indicate that expression of periostin isoforms is regulated both - developmentally and organ-specifically. Whereas the full-length variant may be associated with fundamental processes common to multiple tissues, alternatively spliced isoforms likely reflect specialized roles in tissue-specific developmental programs. However, a comprehensive understanding of periostin isoform function will require systematic analysis across specific tissue-types and developmental stages using standardized approaches. It is important to note that comparisons of periostin isoform expression should be made within the same species, and that differences between *in vitro* models and native tissues must be carefully considered, as alternative splicing is a highly regulated and complex process. One isoform of particular interest, designated periostin-L or the “lung type” of periostin ($\Delta^{17,18,21}$), has been identified in cultured MRC-5 cells (human fetal lung fibroblast line) following treatment with IL-4. Functional studies have shown that periostin-L binds extracellular matrix proteins, such as fibronectin, tenascin-C, and collagen V, in a dose-dependent manner, but not collagens I and III. Remarkably, periostin in mutant form that lacks entire C-terminal domain demonstrated even stronger binding to these partners, suggesting that the FAS1 (fasciclin I) domains are responsible for mediating primary interactions, while the C-terminal region modulates binding affinity [25]. In a set of experiments using cultured human cardiac fibroblasts, the TGF-β1 pathway was used to stimulate periostin expression. The treatment with antibody specifically targeting exon 17 that followed stimulation resulted in significant, dose-dependent reduction in cell viability, indicating that exon 17-containing periostin isoforms play a pro-survival role in these cells [3]. Together, these findings highlight that periostin isoforms can vary not only in tissue distribution but also in functional properties, emphasizing the importance of considering both isoform-specific expression and molecular interactions when interpreting developmental roles. They are tightly regulated during development, with both spatial and temporal specificity reflecting their diverse functional roles. Importantly,

many of these regulatory mechanisms, particularly alternative splicing and isoform-specific interactions, remain active beyond development and are frequently dysregulated in pathological conditions, including cancer.

Periostin and cancer

Tumor-specific and nonspecific isoforms. The presence and functional relevance of periostin isoforms have been investigated across a wide range of malignancies, including cancers of the kidney, lung, breast, pancreas, thyroid, and brain (Figure 2) [2, 5, 8, 24, 26, 27]. These studies indicate that periostin expression in carcinogenesis is highly context-dependent and varies not only between tumor types but also within cancers originating from the same tissue. One of the key challenges in this field is discrimination between tumor-specific and non-specific periostin isoform(s). Reported differences are not limited to the number and types of detected isoforms but also cover their relative abundance and potential specificity for malignant tissue. The definition of the "healthy" control tissue remains problematic, as many studies rely on adjacent non-neoplastic samples near the tumor margin. Although this tissue is histologically normal, it may already harbor molecular alterations associated with early stages of tumorigenesis, potentially including changes in periostin expression. This can be illustrated in thyroid carcinoma, where multiple periostin isoforms were detected in both tumors and adjacent non-neoplastic tissue, suggesting that periostin presence alone may not be sufficient to drive carcinogenesis [27]. In contrast, studies in renal cell carcinoma have demonstrated differential expression of specific splice variants, rather than strict tumor exclusivity - several isoforms were identified in malignant as well as in matched non-neoplastic tissues, however certain variants showed increased abundance in tumors, indicating tumor-associated regulation of alternative splicing. Six isoform sequences that exhibited different relative abundances in normal, fetal, and malignant renal tissues have been described [23]. Further analysis of periostin alternative splicing in kidney tumors identified three isoforms ($\Delta^{17,21}$; $\Delta^{17,18,21}$ and $\Delta^{17,18,19,21}$) that were present exclusively in adult human renal cell carcinoma and in matched non-neoplastic renal tissue but absent in fetal kidney tissue. Among these three variants, $\Delta^{17,18,19,21}$ was more frequently expressed in cancerous tissue compared to the corresponding non-neoplastic samples [8]. These findings suggest that relative isoform expression, rather than just their presence, may have greater biological and clinical relevance.

Interestingly, in contrast to other neoplastic diseases, where cancer-specific periostin variants usually lack one or more exons in the C-terminal region, glioblastoma represents a distinct case with the full-length periostin isoform being specifically associated with the disease. This variant is strongly expressed in glioblastoma cells but largely absent in low-grade gliomas and normal brain tissue, and its expression is restricted to tumor cells rather than the surrounding microenvironment [26].

All in all, these observations underline the complexity of periostin isoform expression in cancer, where both qualitative and quantitative differences contribute to disease-specific patterns. Rather than acting as universally tumor-specific markers, periostin isoforms appear to reflect context-dependent changes in alternative splicing, with potential implications for tumor biology, diagnosis, and therapeutic targeting.

Functional role of periostin in cancer: isoform-specific targeting (exon 17 and exon 21). The role of periostin in cancer could be viewed through the "hallmarks of cancer" concept introduced by Hanahan and Weinberg. Originally, these hallmarks included self-sufficiency in growth signals, resistance to anti-growth signals, invasion and metastasis, limitless replication, sustained angiogenesis, and evasion of apoptosis [28]. This framework was later expanded to include altered metabolism and immune evasion, together with enabling traits [29]. Latest updates further incorporate epigenetic reprogramming, phenotypic plasticity, senescence, and the influence of the microbiome [30]. Several of the hallmark-associated processes have been linked to periostin in previous studies and are discussed in the following sections. Accumulating evidence further suggests that these functions are not uniform but are mediated by distinct periostin isoforms, especially those that involve exons 17 and 21.

A considerable amount of work, particularly in breast cancer models, indicates that periostin variants containing exon 17 contribute foremostly to tumor cell–intrinsic properties. Targeting this region with exon 17-specific antibodies inhibited periostin-induced detachment, proliferation, migration, and invasion of cancer cells *in vitro*, and suppresses primary tumor growth and lung metastasis *in vivo* [2]. Inhibition of primary tumor growth, along with a reduction of lung metastasis, was reported also in the MDA-MB-231 (human triple-negative breast cancer) xenograft model after the treatment with a periostin exon 17 antibodies. This effect was observed even in the 4T1 implanted model of periostin exon 17 KO mice, and lung metastasis was similarly reduced using a morpholino antisense oligonucleotide that skips exon 17 of periostin [31]. Mechanistically, exon 17 appears to shape interactions with binding partners such as Wnt3a, as periostin containing exon 17 is capable of binding this ligand, whereas exon 17-deficient variants show reduced interaction [32]. These findings suggest that presence of exon 17 may influence downstream signaling pathways relevant to tumor progression.

In contrast, periostin variants that contain exon 21 are more strongly associated with the tumor microenvironment, particularly stromal compartments. These isoforms are predominantly expressed by cancer-associated fibroblasts and have been linked to the regulation of angiogenesis, inflammation, and immune modulation. Inhibition of exon 21-containing variants in a mouse triple-negative breast cancer model led to the reduction of tumor growth, decrease of pro-angiogenic (ANGPT1, FGF1) and pro-inflammatory factors (CCL2, CXCL13, IL6, IL1B) expression [6], and limited the accumulation of tumor-supportive M2 macrophages [4]. Moreover, targeting exon 21 increased the efficacy of chemotherapeutic agents such as paclitaxel, suppressed tumor relapse, and reduced mesenchymal-like tumor cell populations [33], suggesting a role in treatment resistance. *In vitro* experiments with Δ^{17} periostin variant-transfected HSC-3 cells resulted in increased cell viability under stress conditions and enhanced chemoresistance. In an HSC-3 xenograft model, co-treatment of an exon 21-specific periostin antibody with cisplatin reduced tumor size more effectively than sole cisplatin. The idea behind the targeting this specific region lies in the observation that periostin variants containing exon 21 were among the most prevalent isoforms detected in patient tumor tissues, and their expression levels correlated with disease progression, while fibroblasts in the tumor-associated stroma were considered a major source of periostin [34].

The functional importance of exon 21 extends beyond breast cancer. In pancreatic ductal adenocarcinoma, cancer-associated fibroblasts have been identified as the primary source of periostin which secrete exon 21-containing variants that contribute to extracellular matrix remodeling, production of cytokines, and chemoresistance, partly via interactions with proteins such as HSP70-1 [5]. After eight periostin variants had been described in the tissues from the patients with pancreatic adenocarcinoma (Supplementary Table S1) [35], the authors focused on the four major variants - in addition to the full-length variant including Δ^{17}, Δ^{21} and $\Delta^{17,21}$, with the variant containing exon 21 but lacking exon 17 being the most prevalent in human cancer-associated fibroblasts [5].

Furthermore, in head and neck cancers, periostin isoforms influence epithelial-mesenchymal transition and invasive behavior, with variants such as Δ^{17} and $\Delta^{17,21}$ promoting cellular invasiveness and expression of EMT-associated genes (Supplementary Table S1) [36]. It is noteworthy that alternative exon annotations have been reported, describing variation in exons 17 and 20 instead of 17 and 21, which may reflect differences in exon numbering conventions: two exons located between exons 17 and 21 in some annotations may be counted as a single exon in others. In line with this, recombinant periostin lacking exons 17 and 20 (consistent with the isoforms detected during the screening) increased cell migration, promoted cell survival under stress conditions, induced β1-integrin expression, and activated the AKT/PKB signaling pathway [11].

In models of another type of cancer - lung cancer, exon 17- and 21- deficient periostin variants promoted proliferation, migration, and resistance to apoptosis, and contributed to immune modulation by enhancing M2 macrophage polarization and production of cytokines IL-10 and TGF-β, while these effects were mediated through activation of PI3K/AKT and JNK signaling pathways [37]. Interestingly, all periostin variants identified in patient non-small cell lung cancer tissues lacked exon 17, supporting the biological relevance of these findings [24].

All things considered, functions of periostin in cancer are highly isoform-dependent, with exon 17-associated variants primarily influencing tumor cell behavior, while exon 21-containing isoforms playing a prominent role in tumor microenvironment shaping and therapy response.

Contradictory roles of periostin in cancer. Periostin, however, can also exhibit opposite, tumor-limiting effects. For example, in an S180 sarcoma allograft model, tumors grew significantly faster in periostin-deficient C57BL/6 mice compared to wild-type controls, accompanied by reduced deposition of fibrillar collagens (mainly collagen I and III) and impaired capsule formation in periostin-deficient hosts [12]. These results imply that endogenous periostin can contribute to organization of extracellular matrix and tumor encapsulation, potentially limiting expansion of the tumor in certain contexts. Isoform-specific effects may shelter these contradictory roles. In aforementioned model, the $\Delta^{17,21}$ variant was detected predominantly in tumor tissue, indicating a potential involvement of this particular splice variant in the observed tumor-limiting effect [12]. Supporting this hypothesis, exogenous $\Delta^{17,21}$ periostin derived from human bladder cancer tissue reduced cancer cell invasiveness and suppressed metastatic potential, whilst full-length periostin from healthy tissue had a similar effect. Contrastly, the $\Delta^{17,18,21}$ variant, that is the most abundant variant in human bladder cancer, showed diminished inhibitory activity on invasiveness [10]. Mechanistic

analyses propose that these differences are more likely related to structural alterations in the C-terminal region than to the presence/absence of exon 18 [10].

Overall, these studies show that the role of periostin in carcinogenesis is not universally pro-tumorigenic, but dependent on the specific isoforms expressed, the cellular context, and the surrounding microenvironment. Recognizing this complexity is critical for understanding periostin biology and considering periostin as a therapeutic target.

Conclusion and future perspectives. Although nearly three times the duration of Odysseus' legendary return to Ithaca after the Trojan War has passed since Horiuchi et al. first described periostin splice variants, the precise roles of individual isoforms in cancer remain incompletely understood [7]. The evidence presented here shows that periostin functions are highly isoform- and context- dependent: exon 17-containing variants primarily affect tumor cell-intrinsic properties, while exon 21-containing isoforms shape the tumor microenvironment and modulate response to the therapy. Moreover, in some contexts, periostin may exhibit opposing effects, limiting tumor growth or its invasiveness, highlighting the complexity of its biological roles.

A significant proportion of studies to date have focused on alternative splicing events in the C-terminal region of periostin, assessing the presence or abundance of isoforms using PCR primers targeting this domain or limited sequencing. However, other variants, such as the N-terminal exon 9-skipped isoform (mRNA: NM_001424174, protein: NP_001411103), are not routinely analyzed but may influence overall periostin function. Incorporation of such overlooked isoforms into future studies could refine the quantitative evaluation of periostin expression and clarify isoform-specific roles in both physiological and pathological contexts.

Overall, advancing our understanding of periostin isoforms requires: i) standardized nomenclature and detection methods to allow meaningful comparisons across studies, ii) systematic functional analyses in relevant tumor models, taking into account effects mediated by both tumor cells and microenvironment, and iii) integration of isoform-specific targeting strategies to evaluate therapeutic potential, particularly in cancers where specific variant/s drive progression or resistance to treatment. Recognizing the contextual and isoform-specific complexity of periostin is essential for translation of mechanistic insights into effective clinical strategies.

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Supplementary data are available in the online version of the paper.

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Figure Legends

Figure 1. Schematic representation of individual periostin splice variants, illustrating their exon composition and alternative splicing patterns.

Figure 2. Graphic overview of the involvement of periostin in cancer-related processes across multiple organs, highlighting its broad role in neoplastic diseases.

Table 1. Periostin splice variants and isoforms: (A) in humans and (B) in mice, as annotated in the NCBI and Ensembl databases.

A	NCBI				Ensembl		
	Transcript	Length (nt)	Protein	Length (aa)	Gene	Transcript ID	Name
Full length	NM_006475	3301	NP_006466	836	ENSG00000133110	ENST00000379747	POSTN-203
$\Delta^{17,18}$	NM_001135934	3130	NP_001129406	779		ENST00000379742	POSTN-201
$\Delta^{17,21}$	NM_001135935	3136	NP_001129407	781		ENST00000541179	POSTN-209
$\Delta^{17,18,21}$	NM_001135936	3046	NP_001129408	751		ENST00000855908	POSTN-211
Δ^{17}	NM_001286665	3220	NP_001273594	809		ENST00000379743	POSTN-202
$\Delta^{17,18,19}$	NM_001286666	3040	NP_001273595	749		ENST00000541481	POSTN-210
$\Delta^{17,18,19,21}$	NM_001286667	2956	NP_001273596	721		ENST00000855909	POSTN-212
Δ^{21}	NM_001330517	3217	NP_001317446	808		ENST00000379749	POSTN-204
Δ^{18}	NM_001424172	3211	NP_001411101	806		ENST00000855910	POSTN-213
$\Delta^{18,21}$	NM_001424173	3127	NP_001411102	778		ENST00000855912	POSTN-215
$\Delta^{9,17,21}$	NM_001424174	3001	NP_001411103	736			
$\Delta^{20,21}$							
$\Delta^{17,20,21}$							
$\Delta^{18,19}$						ENST00000953169	POSTN-219
$\Delta^{17,20}$							

B	NCBI				Ensembl		
	Transcript	Length (nt)	Protein	Length (aa)	Gene	Transcript ID	Name
Full length	NM_001368678	3406	NP_001355607	838	ENSMUSG00000027750	ENSMUST00000081564	Postn-202
$\Delta^{17,21}$	NM_001198766	3130	NP_001185695	783		ENSMUST00000117373	Postn-204
~~$\Delta\Delta^{17}$~~	NM_015784	3214	NP_056599	811		ENSMUST00000073012	Postn-201
Δ^{21}	NM_001198765	3211	NP_001185694	810		ENSMUST00000107985	Postn-203
$\Delta^{20,21}$	NM_001313898	3133	NP_001300827	784			
$\Delta^{17,20,21}$	NM_001313899	3052	NP_001300828	757			

Fig. 1 Download full resolution image

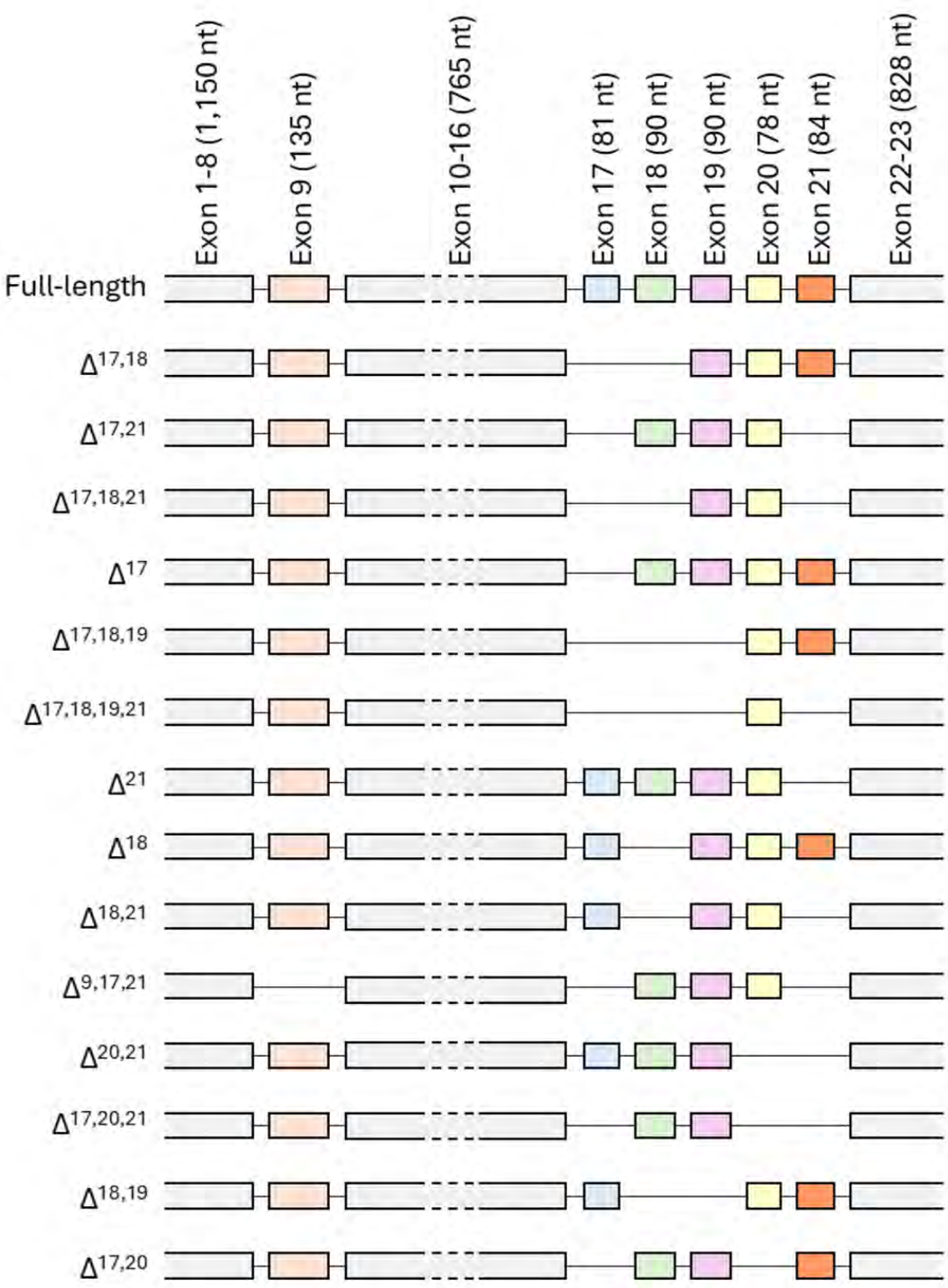

Fig. 2 Download full resolution image