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AFP, and AFP-Derived Growth Inhibitory Peptide; The Longest Unrevealed Secret in Human Pregnancy: A Commentary

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Abstract

Prior to the discovery of a newly detected protein in the serum of fetuses, the existence of a protein later named alpha-fetoprotein (AFP) was not known to scientists in the biomedical world. The fetal AFP molecule was discovered in 1956 and became the first ever reported malignancy biomarker for liver cancer (i.e., hepatoma). In subsequent years, AFP was found to be a pregnancy biomarker for screening birth defects for anencephaly and spina bifida. The presence of AFP in fetal serum led to further biophysical studies of the fetal protein's primary, secondary, tertiary structure. Subsequent studies involving a conformational change in the molecular form of AFP allowed for the discovery of a transformational form of AFP termed "Transformed AFP." The discovery and use of a conformational changed AFP molecule further led to the existence of a hidden and buried 34-amino acid peptide stretch on the third domain of AFP. This peptide segment was termed the "Growth Inhibitory Peptide" and was found to be located withing the transformed AFP polypeptide itself. Thus, the newly discovered triad of the above biomarkers unveiled the previously unknown existence of two more structural entities associated with the AFP molecule that emerged as biomarkers for liver cancer and are involved in all three trimesters of human pregnancy.

Keywords: Alpha-fetoprotein; DNA repair; Bone marrow; Cell cycle; Stem cells; Chromosome instability; Peptides; Breast cancer

Introduction

The Full-Length Alpha-fetoprotein Polypeptide

The biomarker triad forms of human alpha-fetoprotein (AFP) are now known to include A) full length AFP, B) transformed AFP, and C) an AFP-derived Growth Inhibitory Peptide (GIP). Each of these forms have only been reported in biomedical literature during the last several decades. The first member of the triad discovered was the human AFP polypeptide which was reported by Bergstrand and Czar in 1956 [1,2]. This finding resulted from a paper electrophoresis migration pattern which revealed an unknown human fetal serum component. However, increased interest in AFP was further peaked following the report of an association of the AFP polypeptide which involved elevated serum levels in patients fetal with liver cancer (hepatomas) [3,4]. Afterwards, from 1973 to 1977, the work of Chard et al. and Brock et al. utilized maternal serum AFP levels to screen for birth defects for the risk assessments of the antenatal detection of anencephaly and spina bifida, and later for Down Syndrome [5,6].

Transformed Alpha-fetoprotein (TAFP)

The second member of the biomarker triad was termed to be "Transformed AFP" (TAFP), which is a molten globule transient form of AFP. This form of AFP resulted from a transformational change which revealed an incompletely folded form of full-length AFP that circulates in the blood of the fetus, placenta, and host mother [7-9]. The TAFP was discovered by Mizejewski et al. in 1983 while researching the effect of estrogen incubation and activation of the AFP molecule [10-12]. Following a series of studies on the estrogen activation of AFP, Mizejewski et al. reported that AFP underwent a molecular conformational change [13]. It had been found that AFP incubated in high molar concentrations of estrogens and/or esterified fatty acids produced a conformational change in the full-length AFP molecule [14]. This transformational change converted the AFP fetal protein into a transient denatured intermediate misfolded form of the full-length AFP polypeptide which had first been described by Uversky et al. [15-17]. Following

a series of biochemical studies of the AFP-induced conformational change, it was revealed that a new form of full-length AFP (TAFP) was present during pregnancy [7-9,18]. This transformed version of AFP has since been employed as a new biomarker for late-term perinatal distress. The use of TAFP in the obstetrical clinic has since been utilized to detect the following pregnancy disorders:

i. fetal growth restriction and intrauterine growth retardation,

ii. threatened preterm labor,

iii. fetal chronic hypoxic stress, and

iv. fetal blood hemodynamic re-distribution [19].

Overall, the presence of elevated TAFP levels in third trimester maternal serum of pregnant women was found useful in the obstetrical clinic to assess multiple perinatal disorders. These late term disorders have since included assessment of fetal well-being, adverse perinatal outcomes, fetal distress, and late-term deterioration of pregnancy conditions [18]. However, the use of TAFP as a biomarker for late trimester distress disorders has yet to reach its full potential in the obstetric clinic.

Table 1: The three members of the alpha-fetoprotein biomarker triad are listed below in 3 sections:

*Indicates mRNA data from both human and rodent AFP

Section 1: Full-length Alpha-fetoprotein (AFP) Structure and Function Relevance to Isoforms, Epitopes, and Micro heterogenic and Conformational Variants		
Alpha-Fetoprotein Forms	Biological Roles, Activities of AFP Forms	References
1) Genetic Variants* (mRNA Variants)	Fetal Liver: 2,2kb, 1.7kb to 50 to 65kD Protein Neonate of AFP Liver: 1.5kb to 49kD Protein Adult Liver: 1.37kb to 37kD Protein Hepatoma Liver: 1.35kb to 37kD Protein	40-42
2) Soluble Forms A. Free forms B. Bound forms	Candidate proteins bound to AFP could include IgG, IgM, actin, TGF-β, and protease inhibitors released by treatment with 0.4 molar KCL	43-44
3) Molecular heterogeneity A. Carbohydrate forms B. pH isoforms	AFP contains carbohydrate moieties in one-to-three N-linked glycans sites which bind lectins (sugar heterogeneity). AFP can be also separated into 2 forms by pH isoelectric focusing chromatography	45-47
4) Antigenic Variants (epitopes)	AFP contains 6 major antigenic determinant sites when used to raise antibodies. A major epitopic site could span 10,000 Daltons. Major epitopic sites number up to 12 epitopes for an average antibody production.	48-50
5) Non-secreted alpha-fetoprotein (CyAFP) cytoplasmic form	A form of AFP derived from a 37kD AFP retained in the cell as a non-secreted form. It is though to dimerize with truncated steroid receptors and transcription factors.	39,40-44,45-47,50,51,53
Section 2: Transformed Alpha-fetoprotein and Variants		
1) Transformed alpha-fetoprotein (TAFP)	A variant forms of intact AFP which has undergone a conformational unfolding to expose a growth inhibitory peptide segment that suppresses fetal growth during pregnancy	10,29,30,31,33,36-38
2) Denatured intermediate forms	A secondary structured form lacking a rigid tertiary structure is referred to as a "molten globule" (MGF) form of the protein; the MGF is considered a slightly denatured protein state.	6-9
3) Conformational variants	Forms of AFP that have undergone a conformational change in their tertiary structure exposing hidden occult sites.	2,7,8,10-12
4) Misfolded protein forms	An unfolded form of AFP due to faulty chaperoning by HSP 70, HSP 90, and Bip molecules during periods of cellular heat shock, oxidative stress, ischemia, and anoxia	55-57,58-61
Section 3: Alpha-fetoprotein derived Peptides, Fragments & Segments		
1) Synthetic AFP peptide segments A. GIPA B. GI PB C. GIPC D. GIP-34, 36 E. LDSYQCT	Peptide fragmented segments isolated form AFP includes a portion from AFP 14-20, namely LDSYQCT involving sugar transport. Also, a segment termed Growth Inhibitory Peptide (GIP) consisting of 3 internal parts GIP-A, B, C which regulates growth and inhibits cancer growth via cell cycle phase arrest and redox control.	4,13,14,51,68-70,72-74,75-81
2) Enzymatic Fragments	Apoptotic related segments displayed 4 major AFP fragments of 38, 32, 23 and 26kD (after hydrolis) from Domains 1 and 3 and from Raji lymphocyte cell cultures.	43,44
3) Cellular adhesion sequences (3-6 amino acids)	Short AFP peptide segments of 3-6 amino acids detected on AFP match to sequences of Laminin-S, fibronectin, Collagen I and V, Laminin-A, and Laminin B1	23,24

*For each of the subject topics 1-6 listed in Table-1, please refer to the references included in the previously published paper "alpha-fetoprotein structure and function: relevance to isoforms, epitopes, and conformation variants", Exp Biol. Med, Volume 226, Issue-5, pages 377-408, 2001
*C-AFP= cancer-liganded Alpha-fetoprotein; TAFP=Transformed Alpha-fetoprotein; GIP= Growth inhibitory Peptide derived from Alpha-fetoprotein

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The Alpha-fetoprotein Derived Growth Inhibitory Peptide (GIP)

The third member of the biomarker triad is the Growth Inhibitory Peptide (GIP), a 34 amino acid peptide fragment derived from full-length AFP. The AFP-derived peptide segment is known for its ability to inhibit cancer cell growth, for the modulation of autoimmune diseases in both man and animals, and as a pregnancy biomarker for fetal birth defects (see previous section) [20,21]. Thus, GIP is reported to be a peptide segment derived from AFP that is exposed from a buried molecular cleft following a transformational change in the AFP molecule, resulting now in the TAFP form [18,19]. The exposed 34 amino acid peptide segment, derived from the third domain of AFP, was discovered by Mizejewski et al. and published in 1996 [22]. The multiple biological activities of GIP, including activation by estrogens, has been reported and published in the biomedical literature from 1998 to 2010 [23-25]. The estrogen and/or fatty acid activation of the full-length AFP polypeptide was further developed into an in vivo bioassay by Mizejewski et al. for the growth regulatory property of AFP [1-4]. It was later reported that GIP peptides were further capable of binding both the estrogen receptor and estrogenic steroids such as estradiol. GIP was also found to suppress breast and other cancers [26]. Thus, GIP was shown to be a promising new class of amphipathic peptides which potentially could be employed as anti-cancer therapeutic agents (Table 1).

Peptides as Cancer Therapeutic Agents

The term "peptide" was first coined by Emil Fischer in 1921 for which his peptide research was later awarded the Nobel Prize for his peptide research accomplishments [27]. Later, Robert Bruce Merrifield in 1955 conceived and developed the first solid phase peptide synthesis method that revolutionized the field of peptide synthesis [28]. He won a Nobel Prize for his peptide research developments in 1984 [29]. Solid phase synthesis is a process by which a peptide is anchored by a covalent attachment to a peptide C-terminal residue to an insoluble polymer for the subsequent assembly and synthesis of peptides. In summary, peptides are generally known to be 50 amino acids or less in length, compared to proteins which can range from 50 to 600 amino acids or more in length. Peptides are not always approved for use by the FDA seemingly due to the such facts as:

A. some peptides can occur as naturally occurring substances.

B. Peptide have less defined safety and efficacy criteria;

C. undergo uncertified manufacturing and standardized conditions, and

D. their less protected intellectual property claims [30,31].

In contrast, however, the multiple favorable conditions for the clinical usage of peptide can encompass

1) short in vivo half-lives,

2) in vivo temporal stability,

3) favorable elimination/degradation conditions;

4) optimal ranges of bioavailability,

5) rapid removal from treatment areas (to avoid sustained or prolonged effects),

6) excellent targeting abilities together with cell membrane penetration, and

7) drug-cargo carrying delivery into cells [32,33].

Some such peptides also share and display characteristics and traits of the antimicrobial amphipathic peptides [34,35]. These later peptides can form pores and transmembrane channels into cell membranes and bind to negatively charged cell surface membranes of cancer cells, but not normal, non-cancer cells.

The Longest Kept Secret Concerning the Triad Biomarkers in Human Pregnancy

The longest kept secret of the biomarker triad during pregnancy can be ascribed to the lack of published knowledge concerning the use and function of the triad biomarkers. Being unknown to all scientists prior to the discovery of HAFP in 1965, knowledge of a previously undetected protein in fetal blood was not known to exist in pregnant mammals including man [1,2]. The discovery of an AFP fetal protein during pregnancy in mammals revealed the presence of an unknown gestational protein which was produced and secreted by the placenta, and fetus, which also circulated in the maternal serum of the pregnant mother. After delivery of the fetus at term pregnancy in the mother and later in the newborn, AFP polypeptide is no longer produced in any adult human being unless the person develops liver cancer. Thus, the biomarker triad is only present in a woman's life for a 9-month period at each pregnancy, and not at all in the life of a male. It was also totally unknown at the time of AFP's discovery that the fetal protein was capable of a molecular transformation into a denatured intermediate molecular form (TAFP); this form would later be used in the clinic as a late term pregnancy biomarker for perinatal distress and fetal well-being [7-9]. After the time of the AFP discovery, it was then determined that the transformed version of AFP (TAFP) could be transformationally induced as a molecular entity in fetal cellular environments containing high concentrations of estrogen and fatty acids [36].

Again, unbeknownst to the scientific community, it was revealed that a 34-mer peptide fragment, derived from AFP, could serve as a growth regulator and inhibitor for cancer growth and proliferation [11-13]. It was the isolated and purified free form of the AFP-derived 34-mer peptide, that possessed special properties that could be harnessed to serve mankind and made available to treat cancer in adults [20]. The free 34- amino acid peptide is not able to be synthesized in the body of any adult person living at

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present. That is, the human body cannot synthesize or produce AFP, TAFP, or GIP except in a woman during pregnancy. However, AFP remains present in the maternal circulation of pregnancy for only 2-3 weeks postpartum, then the gene for AFP is methylated and turned off in the mother [18]. In the newborn, AFP is synthesized in the liver and is present in the neonatal blood vascular system in microgram amounts for 8-9 months following pregnancy [37,38]. AFP liver production is then greatly reduced to small microgram amounts at 8-9 months of age in the infant. After 9-month time, the synthesis and production of liver AFP is further reduced, and AFP blood levels gradually decline to less than 20 ng/ml in the late infant and juvenile stages [39]. Thereafter, the AFP blood levels in adults are eventually reduced to low nanogram amounts (5-10ng/mL).

Concluding Statements

It can be deduced from the forgoing discussions that the three fetal biomarker components during pregnancy had remained concealed and buried for long periods of time without scientific detection until the mid-nineteenth century. Although AFP was discovered and reported in the mid-1950s, TAFP and GIP remained undetected for another 20 years [9,11]. Thus, for many centuries of the modern scientific age, fetal AFP polypeptide and its derivatives (i.e., TAFP and GIP) remained undetected being only in fetal blood and amniotic fluid of pregnant women. This undetected presence was due to lack of knowledge of conformational changes in proteins that had not yet been fully developed and studied prior to the discovery of modern-day protein technologies. The techniques developed were as follows:

1) fluorescence resonance energy transfer (FRET),

2) Second harmonic probe orientation,

3) surface plasmin resonance, and

4) circular dichroism.

In summary, structural engineering knowledge of protein conformational changes are now known to be necessary for designing new proteins and peptides with specific features that have yet to be discovered. The above methods later into being and offer certain various advantages, such as measuring inter-atomic distances in real time, probing conformational dynamics at the molecular level. At present, such conformational change measurements have now been developed and applied to various diseases; thus, understanding these changes can lead to better diagnostic and therapeutic modalities and strategies [40-50].

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Conflicts of Interest

The author declares there are no known conflicts of interest in the preparation of this manuscript.

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Interest

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