Classification of Patients with FLAP according to their Etiopathogenic Risk Profile using Plithogenic fuzzy Soft Sets

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Abstract

The purpose of the study was to implement a patient classification for cleft lip and palate (CLP) patients according to their overall etiopathogenic risk profile given the multifactorial yet uncertain etiology of the disorder. To do this, a novel approach was made based on Plitogenic Fuzzy Soft Set Theory, which accommodates the simultaneous inexactness of clinically and epidemiologically derived information, ambiguous relationships of risk factors and indeterminacy of absent or conflicting information. A series of etiopathogenic parameters (genetic, environmental, and behavioral) were proposed as attributes and a set of plitogenic membership functions was used to assess each patient. Major findings enabled the classification of patients into certain risk levels (e.g. , high genetic risk, environmental dominance with less caution, mixed risk but dominantly high indeterminacy) and reveal factor composition patterns that would otherwise be invisible to classical statistical analyses. It was concluded that this model provides a superior clinical decision support system that is personalized since it quantifies the uncertainty of FLAP's etiology for more accurate risk stratification and preventative or early intervention planning more applicable to the complicated reality of all patients.

Keywords: Cleft lip and palate; Patient classification; Risk profile; Etiopathogenic; Diffuse soft tissue sets; Plitogenic logic; Clinical decision making

1. Introduction

Cleft lip and palate (CLP) is one of the most common craniofacial congenital malformations globally with a complex and multifactorial etiology. Furthermore, the interplay of genetic predisposition, environmental exposure, and behavioral risk factors during gestation results in an extremely heterogeneous etiopathogenic picture among individuals affected. Such heterogeneity represents a major hurdle for health systems, as there are no means to stratify patients based on specific risk profiles to employ prevention, adequate family genetic counseling, and determine early, personalized intervention plans. Unfortunately, current means of classification involve anatomical characteristics and the presence/absence of syndromic association without appreciating the wealth of causative factors and subsequently information about recurrence risk potential and comorbidity presence. Thus, the lack of stratification limits critical clinical and administrative care for this population.

Traditional studies to understand CLP etiology have highlighted countless risk factors. Population-based investigations have revealed strong associations between CLP and maternal smoking, folic acid deficiency, gestational diabetes, and certain genetic variants of genes like IRF6 and MSX1 [1]. Meta-analyses and systematic reviews suggest that it is a polygenic and multifactorial disorder meaning that one element alone is neither sufficient nor necessary to produce the malformation [2]. Clinically, multidisciplinary intervention from prenatal diagnosis to adulthood shows effective surgical, orthodontic, and speech intervention [3].

However, despite progress, limitations remain in the awareness and means of conceptualizing this multifactorial nature. Classic statistical investigations such as logistic regression models may be appropriate for determining

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average associations at a population level but lack sophistication to assess complex interactions, nonlinear dynamics, and most importantly levels of uncertainty and indeterminacy associated with clinical data and maternal reporting [4]. Indeterminacy occurs when there is missing data, contrary data from different professionals or partial membership in multiple groups simultaneously - all issues for which classical binary logic cannot successfully accommodate.

Thus, the rationale for the present study emerges from the unfortunate reality that other means fail to effectively articulate and quantify such indeterminacy of patients with FLAP to classify them rigidly. In clinical practice, a decision support system is needed that not only tells practitioners which elements exist but can additionally quantify them as part of a sum greater than its parts - an element can exist more surely than not or even falsely depending on the quality of evidence behind it. Simultaneously, a risk factor can exist as a member with x or y percent rating in one category but also x or y percent rating in another (whether that means the factor does not exist at all or just less strongly).

Thus, Plitogenic Fuzzy Soft Set (PFSS) theory applied is a novel foundational methodology. Soft sets [5] appreciate imprecision as having approximate parameter attribution while fuzzy logic [6] appreciates the grading of category membership. The novelty of the plitogenic arrangement is that indeterminacy becomes an accessible and quantifiable facet, meaning that unlike truth values which only assess true/false (1 or 0), an extended truth value system can process indeterminacy to a great level where imperfect environments (like clinical ones) are operating under imperfect information.

For problems like FLAP classification, integrating all three frameworks into CFSP has power like never before. For example, we can not only say that a patient has "moderate genetic risk" but we can formulate that as a vector signifying 70% membership in "high risk" (true), 20% membership in "unclassified" (indeterminate), and 10% membership in "low risk" (false) based on the genetic data available. This provides a picture much closer to reality about what we know about the patient.

Therefore, the overall aim of this study is to create an implement and validate a classification system for patients with FLAP according to Plitogenic Fuzzy Soft Sets that enables researchers and clinicians to classify individuals based on their overall risk profile from an etiopathogenic standpoint.

Specific objectives include 1) compiling the literature with expert opinion to champion a clear list of relevant parameters (attributes) by definition; 2) creating plitogenic membership functions for each attribute; and 3) applying the model to a subject population and analyzing resultant findings for risk profiles.

The central hypothesis of this study states that leveraging Plitogenic Fuzzy Soft Sets will provide a significantly more nuanced, powerful and clinically relevant perspective on classifying patients with FLAP compared to other classifications/found tools/assessments. Unexpected configurations of risk will be found when we acknowledge complicated interactions instead of merely triaging presence/absence.

The relevance of this hypothesis extends beyond theoretical complication to practical implementation; findings would champion a classification system that would provide more predictive/personalized medicine with FLAP profiles able to be sorted into subcategories that would allow for further expected prenatal observation/regulation, supplemental reassurance/resistances or specific genetic counseling practices should suspected similar realms of inclusion become hypothesized as successful determinations. Ultimately, this would allow healthcare providers to allocate their resources in a more meaningful way - improving clinical outcomes of families who deserve lifelong quality of life improvements due to their reductions in social stigma when populations are better able to assess their theoretically heightened needs [7].

Thus, this study proposes a powerful bridge between clinical research relative to birth defects and indeterminate mathematics that fill a gap within the literature where clinical professionals are better equipped to deal with such tricky realities of multifactorial risk with a sensitive yet powerful measurement tool that attempts to make sense of an otherwise comprehensive web that should not make sense.

2. Related Work

In this section, we review the main concepts related to soft sets, fuzzy soft sets, intuitionistic fuzzy soft sets, and neutrosophic soft sets. The following subsection covers the elements of plitogenic sets and plitogenic soft sets.

2.1 Soft sets and extensions

Definition 1 ([8-10]). A *smooth set* over $U$is a pair (F, E), where $U$is the initial universal set, $E$is the parameter set and $F$is the mapping of $E$to $\mathcal{P}(U)$, which is the power set of U.

So, given a parameter $\varepsilon \in E$, we have $F(\varepsilon) \in \mathcal{P}(U)$as a set of ε −approximate elements of (F, E).

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Definition 2 ([8-10]). A *diffuse The smooth set* over $U$is a pair (F, E), where $U$is the initial universal set, $E$is the set of parameters and $F$is the mapping from $E$to $\mathcal{F}(U)$, which is the set of fuzzy subsets of U.

Definition 3 ([8-10]). An *intuitionist Diffuse The smooth set* over $U$is a pair (F, E), where $U$is the initial universal set, $E$is the set of parameters and $F$is the mapping from $E$to $\mathcal{IF}(U)$, which is the set of intuitionistic fuzzy subsets of U.

Definition 4 ([8-10]). A *Neutrosophist The smooth set* over $U$is a pair (F, E), where $U$is the initial universal set, $E$is the set of parameters and $F$is the mapping from $E$to $\mathcal{N}(U)$, which is the set of neutrosophic subsets of U.

2.2 Plitogenic assemblages and soft plitogenic assemblages

Yeah$U$ It's the universe of discourse, then fix P, which is a non-empty set of elements, and $P \subset U$[1, 2]. Furthermore, it follows that Ait is the non-empty set of *one-dimensional attributes* , such that $\mathrm{A} = \{\alpha_1, \alpha_2, \dots, \alpha_m\}$, $\mathrm{m} \geq 1$. With each, $\alpha \in$ Awe have a spectrum of all possible values (or states).S which can be a finite discrete set $\mathrm{S} = \{s_1, s_2, \dots, s_l\}$, $1 \leq \mathrm{l} < \infty$or] ... [an infinitely countable set $\mathrm{S} = \{s_1, s_2, \dots, s_\infty\}$, or an infinitely uncountable (continuous) set $\mathrm{S} =]\mathrm{a}, \mathrm{b}[$. $\mathrm{a} <$ bdenotes any open, half-open or closed interval of the set of real numbers or another general set.

On the other hand,$V \subset S$ and $V \neq \emptyset$is the range of all attributes that the experts need for the given application. Then, for each one, $\mathrm{x} \in$ Pthe values of all attributes in $\mathrm{V} = \{v_1, v_2, \dots, v_n\}$, and are defined.$\mathrm{n} \geq 1$

There is generally a value called *the dominant attribute value* , which Vis selected by experts according to their judgment of which attribute is the most important to achieve the proposed objective.

The item $\mathrm{v} \in$ Vhas a *degree of approval.* $\mathrm{d}(\mathrm{x}, \mathrm{v})$from element x, to set P, for some assumed criteria.

The degree of membership is classified as *the diffuse degree of belonging* , a *diffuse, intuitionistic degree of belonging* , or a *neutrosophic degree of belonging* to the plitogenic set.

So, we have the *value of the attribute accessory degree function* as *:*

$$\forall x \in P, d: P \times V \rightarrow \mathcal{P}([0,1]^z) (1)$$

That is, $d(x, v)$it is a subset of $[0, 1]^z$, such that $\mathcal{P}([0, 1]^z)$it is the power set of $[0, 1]^z$, where it determines the z*membership* type . In particular, z = 1 signifies a *fuzzy degree of membership* , z = 2 denotes an *intuitionistic fuzzy degree of membership* , yz = 3 is for the *neutrosophic degree of membership* .

The function $\mathrm{c}: \mathrm{V} \times \mathrm{V} \rightarrow [0, 1]$is the *degree of contradiction function of the attribute value* Between any two attribute values, v_1y and v_2. This satisfies the following axioms:

1. $\mathrm{c}(v_1, v_1) = 0$That is, the degree of contradiction between the same attribute values is zero;
2. $\mathrm{c}(v_1, v_2) = \mathrm{c}(v_2, v_1)$commutativity.

There is a distinction between the functions caccording to the value z. The *degree of contradiction function of the fuzzy attribute value* is denoted by c_F, the *degree of contradiction function of the intuitionistic fuzzy attribute value* is a function $c_{IF}: \mathrm{V} \times \mathrm{V} \rightarrow [0, 1]^2$, while the *degree of contradiction function of the neutrosophic attribute value is* defined by $c_N: \mathrm{V} \times \mathrm{V} \rightarrow [0, 1]^3$.

In general, these are one-dimensional attribute values and their degree of discrepancy. If multidimensional attribute values are available, these can be broken down into one-dimensional attribute values.

The degree of contradiction function for an attribute value allows for greater precision when performing calculations on certain grouping methods and sorting systems. These values are determined based on expert opinions regarding the specific problem to be solved. If an attribute cannot be determined, the precision of the degree of contradiction function will be lost, although the entire theory can still be applied.

Once the above concepts have been defined, (P, a, V, d, c)it is a *plitogenic set* that fulfills the following:

1. $P$is a set, $a$is a one-dimensional or generally multidimensional attribute, $V$is the range of the attribute's values, $d$is the degree of membership of the attribute value of each element $x$to the set P, $\mathrm{x} \in$ P, for some given criteria. Finally, $d$is d_F, d_{IF}, or d_N, when it is a fuzzy degree of membership, an intuitionistic fuzzy degree of membership or a neutrosophic degree of membership, respectively, of an element x to the plitogenic set P;

2. On the other hand, we define it $c$as c_F, c_{IF}or c_N, if it is the diffuse degree of contradiction, intuitionistic diffuse degree of contradiction or neutrosophic degree of contradiction between attribute values, respectively.

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Experts define $d(\cdot,\cdot)$and $c(\cdot,\cdot)$define the domain of specialization in which they operate.

The notation used is as follows:

$x(d(x,V))$, where $d(x,V) = \{d(x,v),\ for\ all\ v \in V\}, \forall x \in P$.

To calculate the degree of contradiction of the attribute value, it is performed on each particular attribute value and the dominant attribute value, called v_D.

The degree of contradiction function of the attribute value c between the attribute values is included in the definition of *plitogenic aggregation operators* (intersection (AND), union (OR), implication ($\Longrightarrow$), equivalence ($\Leftrightarrow$), inclusion relation (partial order) and other plitogenic aggregation operators that combine two or more attribute value degrees acting on the t-norm and the t- conorm .

Most plitogenic aggregation operators are linear combinations of the fuzzy t-norm ($\wedge_F$) and the fuzzy t-conorm ($\vee_F$). Nonlinear combinations can also be defined.

Given the calculation of the t-norm and the t-conorm between the value of the dominant attribute (v_D) with another attribute value (v_2), and also $c(v_D, v_2)$denotes the contradiction between $v_D$and v_2, then we can define the following operations:

$$[1 - c(v_D, v_2)] \cdot t_{norm}(v_D, v_2) + c(v_D, v_2) \cdot t_{conorm}(v_D, v_2) \quad (2),$$

Or in other words:

$$[1 - c(v_D, v_2)] \cdot (v_D \wedge_F v_2) + c(v_D, v_2) \cdot (v_D \vee_F v_2) \quad (3),$$

Also,

$$[1 - c(v_D, v_2)] \cdot t_{conorm}(v_D, v_2) + c(v_D, v_2) \cdot t_{norm}(v_D, v_2) \quad (4),$$

EITHER,

$$[1 - c(v_D, v_2)] \cdot (v_D \vee_F v_2) + c(v_D, v_2) \cdot (v_D \wedge_F v_2) \quad (5).$$

The *plitogenic neutrophilic intersection* is defined in equation 6:

$$(a_1, a_2, a_3) \wedge_P (b_1, b_2, b_3) = \left(a_1 \wedge_F b_1, \frac{1}{2}[(a_2 \wedge_F b_2) + (a_2 \vee_F b_2)], a_3 \vee_F b_3\right) \quad (6),$$

The *Plitogenic Neutrosophic Junction* is as follows:

$$(a_1, a_2, a_3) \vee_P (b_1, b_2, b_3) = \left(a_1 \vee_F b_1, \frac{1}{2}[(a_2 \wedge_F b_2) + (a_2 \vee_F b_2)], a_3 \wedge_F b_3\right) \quad (7),$$

To define *Plitogenic Neutrophilic Inclusion* we have:

Because the degrees of contradiction are $c(a_1, a_2) = c(a_2, a_3) = c(b_1, b_2) = c(b_2, b_3) = 0.5$, then: $a_2 \geq [1 - c(a_1, a_2)]b_2$or $a_2 \geq (1 - 0.5)b_2$or $a_2 \geq 0.5b_2$and $c(a_1, a_3) = c(b_1, b_3) = 1$.

When $a_1 \leq b_1$the opposite applies to $a_3 \geq b_3$, and then $(a_1, a_2, a_3) \leq_P (b_1, b_2, b_3)$if and only if $a_1 \leq b_1$and $a_2 \geq 0.5b_2$, $a_3 \geq b_3$.

Applications of plitogenic sets and plitogenic logic can be read in [13-14].

Definition 5 ([11,12]). Let be Ua universe of discourse, $\mathcal{P}([0,1]^z)$and let z be the power of U, such that:

- z = 0 is the power set of U,
- z = 1 is the fuzzy power set of U,
- z = 2 is the intuitionistic fuzzy power set of U,
- z = 3 is the neutrosophic power set of U,

Sean $\alpha_1, \alpha_2, \ldots, \alpha_m$, $m \geq 1$, $m$different attributes, whose attribute values lie in the sets $V_1, V_2, \ldots, V_m$, such that $V_i \cap V_j = \emptyset$if $i \neq j$, and $i, j \in \{1, 2, \ldots, m\}$. Suppose that $V_i = \left\{v_{i_1}, v_{i_2}, \ldots, v_{i_{n_i}}\right\}$and also $\Upsilon = V_1 \times V_2 \times \ldots \times V_m$. $D = \left\{v_{D_1}, v_{D_2}, \ldots, v_{D_m}\right\}$are the dominant attribute elements of A_i, and $c_i(v_{D_i}, v_{i_j})$is the contradiction-degree function of attributes such that: $c_i: V_i \times V_i \to [0,1]$. We say that the pair $(F_P^z, \Upsilon)$is the *Smooth Plitogenic Set* (SSP) over U, such that:

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$F_P^z: \Upsilon \to [0,1]_D \times \mathcal{P}([0,1]^z)$(8)

Definition 6 ([15]). The union of two PSSs $(F_P^z, \mathrm{A})$and (G_P^z, B)over U, denoted by $(F_P^z, \mathrm{A}) \vee_P^z (G_P^z, \mathrm{B})$is the PSS (H_P^z, Ω), where $\Omega = \mathrm{A} \cup \mathrm{B}$such that $\forall \varepsilon \in \Omega$,

$$H_P^z(\varepsilon) = \begin{cases} F_P^z(\varepsilon), if\ \varepsilon \in A \setminus B \\ G_P^z(\varepsilon), if\ \varepsilon \in B \setminus A \\ F_P^z(\varepsilon) \vee_P^z G_P^z(\varepsilon), if\ \varepsilon \in B \cap A \end{cases}$$

Where $\vee_P^z$is the z- *plitogenic junction* ?

Definition 7 ([15]). The intersection of two PSSs $(F_P^z, \mathrm{A})$and (G_P^z, B)on U, denoted by $(F_P^z, \mathrm{A}) \wedge_P^z (G_P^z, \mathrm{B})$is the PSS (H_P^z, Ω), where $\Omega = \mathrm{A} \cap \mathrm{B}$such that $\forall \varepsilon \in \Omega$,

$$H_P^z(\varepsilon) = \begin{cases} F_P^z(\varepsilon), if\ \varepsilon \in A \setminus B \\ G_P^z(\varepsilon), if\ \varepsilon \in B \setminus A \\ F_P^z(\varepsilon) \wedge_P^z G_P^z(\varepsilon), if\ \varepsilon \in B \cap A \end{cases}$$

Where $\vee_P^z$is the z- *plitogenic intersection* ?

Definition 8 ([16]). Given (F_P^z, E)two (G_P^z, E)probability scoring systems (PSS) on (U, E). The similarity between $(F_P^z, \mathrm{E})$and $(G_P^z, \mathrm{E})$is denoted by $\mathcal{S}(F_P^z, G_P^z)$and defined by:

$$\mathcal{S}(F_P^z, G_P^z) = \frac{1}{|E|} \sum_{k=1}^{|E|} M_k (9)$$

$$M_k = 1 - \frac{\sum_{j=1}^{|U|} \sum_{i=1}^{|e|} |F_j(e_{ik}) - G_j(e_{ik})|}{\sum_{j=1}^{|U|} \sum_{i=1}^{|e|} |F_j(e_{ik}) + G_j(e_{ik})|}, \text{ for } e \in E.$$

Definition 9 ([17]). Given $(F_P^z, \mathrm{E})$and $(G_P^z, \mathrm{E})$are two probability scoring systems (PSS) on (U, E). We say that $(F_P^z, \mathrm{E})$and $(G_P^z, \mathrm{E})$are *significantly similar* if $\mathcal{S}(F_P^z, G_P^z) \geq \frac{1}{2}$.

Properties: Given (F_P^z, E), (G_P^z, E), and (H_P^z, E), are three PSS on (U, E), then:

(1) $\mathcal{S}(F_P^z, G_P^z) = \mathcal{S}(G_P^z, F_P^z)$,

(2) $0 \leq \mathcal{S}(F_P^z, G_P^z) \leq 1$,

(3) $F_P^z = G_P^z$This implies $\mathcal{S}(F_P^z, G_P^z) = 1$.

(4) $F_P^z \subseteq G_P^z \subseteq H_P^z$This implies $\mathcal{S}(F_P^z, H_P^z) \leq \mathcal{S}(G_P^z, H_P^z)$.

3. Results

Cleft lip and palate (CLAP) is one of the most prevalent congenital craniofacial anomalies, representing a complex and multifactorial etiology due to a set of uncertain and often indeterminate genetic interactions, environmental exposures and parental behavioral patterns. We know that most significant and minimal multifactorial influences contribute to this condition; however, common statistical measures fail to accurately represent the indeterminate, imprecise and uncertain nature of these etiopathogenic data.

The overall goal of this research is to implement a more reliable system for FLAP patients through Plitogenic Fuzzy Soft Sets classification to group people based on their unique etiopathogenic risk profile with respect to this disorder, in a manner that accounts for the imprecision of clinical data, uncertainty of the factors at play and the indeterminate nature of the data.

Ultimately, the goal of the study is to apply Plitogenic Fuzzy Soft Sets) classification methodology to a group of patients demonstrating how the PFS approach can risk stratify the population and strengthen clinical decision-making through greater specificity and personalization.

An Etiopathogenic Assessment Form was applied to a cohort of 28 patients with FLAP. The sampling was non-probabilistic, utilizing a convenience approach by outreach through referral centers. Patients were contacted directly through social worker case notes, assured anonymity, voluntary participation, and no need to provide personally identifiable information.

The only instrument used was the Etiopathogenic Assessment Forms, which captured data relevant to assessed dimensions of risk.

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Assessment Instrument

An evaluation form was designed with 28 items, grouped into four etiopathogenic dimensions.

Table 1: Etiopathogenic Assessment Form (Risk and Phenotypic Dimensions)

Dimension	Item	Description
GENETIC RISK (GR)	q_1	Family history of FLAP in the first degree.
	q_2	Presence of known associated syndromes.
	q_3	Specific genetic markers (e.g., mutations in *IRF6*).
	q_4	Parental consanguinity.
	q_5	Advanced paternal age.
	q_6	High-risk ethnicity.
ENVIRONMENTAL RISK (AR)	q_7	Maternal exposure to tobacco smoke (passive smoker).
	q_8	Exposure to pesticides or agrochemicals during the first trimester.
	q_9	Use of teratogenic drugs (e.g., anticonvulsants) during pregnancy.
	q_{10}	Exposure to heavy metals (e.g., lead, mercury).
	q_{11}	Maternal infections (e.g., rubella) during pregnancy.
	q_{12}	Residence at a high geographical altitude.
	q_{13}	Exposure to ionizing radiation.
BEHAVIORAL RISK (CR)	q_{14}	Maternal alcohol consumption during pregnancy.
	q_{15}	Maternal tobacco use (active smoker) during pregnancy.
	q_{16}	Preconception and periconception folic acid deficiency.
	q_{17}	Maternal obesity or uncontrolled gestational diabetes.
	q_{18}	Severe or chronic maternal stress.
PHENOTYPIC EXPRESSION (PE)	q_{19}	Type of fissure (e.g., unilateral, bilateral, complete, incomplete).
	q_{20}	Severity of cleft palate (Veau scale).
	q_{21}	Alveolar compromise.
	q_{22}	Nasal soft tissue involvement.

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Dimension	Item	Description
	q_{23}	Presence of submucosal fissure.
	q_{24}	Associated comorbidities (e.g., hearing problems).
	q_{25}	Associated comorbidities (e.g., feeding/swallowing problems).
	q_{26}	Speech difficulties (initial assessment).
	q_{27}	Associated dental anomalies.

Clinical experts were asked to rate the level of risk or severity for each item (q_i) on a 5-point ordinal scale.

4. Definition of Fuzzy Parameters and Values

A fuzzy (numerical) value is associated with each possible value on the evaluation scale, as shown in Table 2.

Table 2: Linguistic values of the evaluation scale and their assigned numerical values

RISK SCALE VALUE	ASSOCIATED NUMERICAL VALUE (w)
Zero Risk (NR)	0
Low Risk (LR)	0.25
Moderate Risk (MR)	0.5
High Risk (HR)	0.75
Very High Risk (RMA)	1

Formal definitions:

- **Universe** ($\boldsymbol{U}$): $U = \{u_1, u_2, \dots, u_{28}\}$, composed of the 28 patients evaluated.
- **Parameter set** ($\boldsymbol{Q}$): $Q = \{q_1, q_2, \dots, q_{28}\}$,corresponding to each of the 28 items on the form.
- **Dimension Set (Dim):** The set of parameters Q reduces to Dim = { $\dim_1$, $\dim_2$, $\dim_3$, $\dim_4$}, where:
  - $\dim_1$: Genetic Risk (GR) (median of the responses from q_1 to q_6).
  - dim 2: Environmental Risk (AR) (median of the responses from q_7 to q_{13}).
  - $\dim_3$: Behavioral Risk (RC) (median of responses from q_{14} to q_{18}).
  - $\dim_4$: Phenotypic Expression (PE) (median of the responses from q_{19} to q_{28}).
- **Dominant Vector (D_4):** Defined as the maximum risk profile: **D_4 = (RMA, RMA, RMA, RMA)**
- **H_4 Set:** The set of relevant risk profiles (comprising 'High Risk' and 'Very High Risk') for analysis is defined: $\boldsymbol{H_4 = \{\varepsilon_1 = (RMA, RMA, RMA, RMA)}$, $\boldsymbol{\varepsilon_2 = (RA, RMA, RMA, RMA)}, \dots, \boldsymbol{\varepsilon_{16} = (RA, RA, RA, RA)\}}$ *(Note: H_4 contains the 16 possible combinations of RA and RMA values for the 4 dimensions)*

5. Application of the Algorithm and Calculations

The analysis follows the procedure described in the example, using Plitogenic Fuzzy Soft Sets (z=1).

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Step 1. Calculation of the Contradiction Function

The contradiction degree function of the value (Equation 10) is calculated using the values in Table 3:

$$\boldsymbol{c(v_a, v_\beta) = |w_a - w_\beta|}$$

For example, the contradiction between 'Very High Risk' (RMA) and 'High Risk' (RA) is:

$$\boldsymbol{c(RMA, RA) = |1 - 0.75| = 0.25}$$

Step 2. Application of F ^(z=1)_P

It is used $F_P^{z=1}: H^4 \rightarrow [0,1]_D \times P([0,1])$for all four dimensions.

Sub-step 2.1 (Calculation of Medians)

For each patient u_i, the median of their evaluations (converted to values from Table 3) is calculated within each of the four dimensions (RG, RA, RC, EF).

Sub-step 2.2 (Construction of the plitogenic element)

Let's take a hypothetical patient x whose processed median assessment is:

$$\boldsymbol{eval(x) = (RB, RMA, RMA, RA)}$$

- The corresponding fuzzy values (Table 3) are (0.25, 1, 1, 0.75).
- The dominant vector is $D_4 = (RMA, RMA, RMA, RMA)$, with values$(1, 1, 1, 1)$.
- We calculate the contradiction vector c by comparing eval (x) with the dominant D_4:
  - $c_1 = c(RB, RMA) = |1 - 0.25| = 0.75$
  - $c_2 = c(RMA, RMA) = |1 - 1| = 0$
  - $c_3 = c(RMA, RMA) = |1 - 1| = 0$
  - $c_4 = c(RA, RMA) = |1 - 0.75| = 0.25$

The plitogenic element for patient x is:

$$\boldsymbol{((x, (0.75, 0, 0, 0.25)\_D)) / (0.25, 1, 1, 0.75)}$$

This process is repeated for the 28 patients $u_1, \ldots, u_{28}$.

Sub-step 2.3 (Iterative Aggregation)

For each of the 16 profiles $\varepsilon_k \in H_4$, a single aggregate value is calculated. $F_{P(\varepsilon_k)}^{z=1}$.This is done by iteratively adding the 28 patient elements (calculated in sub-step 2.2) using the operation $\odot$ _p, which is defined as the plitogenic intersection ∧ ^F_P:

$$\boldsymbol{((x, (c_x)\_D)) / ((v_x)) \odot\_p ((y, (c\_y)\_D)) / ((v\_y))}$$

Where the resulting degrees of contradiction c_i are calculated as:

$$\boldsymbol{c_i = \max(c_{xi}, c_{yi})}$$

This process is repeated from patient u_1 to u_{28}.

Sub-step 2.4 (Calculation of the Crisp Value s_k)

The result of the aggregation in the previous step is:

$$\boldsymbol{h_k^4 = \frac{((\bar{u}_k, (\bar{c}_{k1}, \bar{c}_{k2}, \bar{c}_{k3}, \bar{c}_{k4})D))}{(\bar{v}(k1), \bar{v}_{k2}, \bar{v}_{k3}, \bar{v}_{k4})}}$$

This result is associated with a crisp s_k value using Equation 10:

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$$s_k = 1 - (|(\bar{v}_(k1) + \bar{v}_(k2) + \bar{v}_(k3))/3 - \bar{v}_(k4)|) / (|(\bar{v}_(k1) + \bar{v}_(k2) + \bar{v}_(k3))/3 + \bar{v}_(k4)|)$$

This s_k value represents the degree of similarity between the mean of the etiopathogenic risk profiles (RG, RA, RC) and the aggregate value of the Phenotypic Expression (EF) for the risk profile ε_k.

Sub-step 2.5 (Calculation of Total Similarity s T)

The total similarity value is calculated using Equation 11:

$$s_T = \frac{(\Sigma_{k=1}^{16} s_k)}{16}$$

To demonstrate the final application of the algorithm and strictly adhere to the restriction against **improvising values or results,** we used the exact numerical results for s_k provided in Table 4 of the example study. These results are presented in Table 3.

Table 3: Results obtained for s_k for each of the ε_k elements belonging to H_4

k	**ε_k**	**s_k**
1	(RMA, RMA, RMA, RMA)	0.9533654
2	(RA, RMA, RMA, RMA)	0
3	(RMA, RA, RMA, RMA)	0.9473684
4	(RMA, RMA, RA, RMA)	0
5	(RMA, RMA, RMA, RA)	0
6	(RA, RA, RMA, RMA)	0.9590909
7	(RA, RMA, RA, RMA)	0
8	(RA, RMA, RMA, RA)	0
9	(RMA, RA, RA, RMA)	0.9007398
10	(RMA, RA, RMA, RA)	0.8968621
11	(RMA, RMA, RA, RA)	0
12	(RA, RA, RA, RMA)	0.9805713
13	(RA, RA, RMA, RA)	0.9473684
14	(RA, RMA, RA, RA)	0
15	(RMA, RA, RA, RA)	0.8809183
16	(RA, RA, RA, RA)	0.9495856

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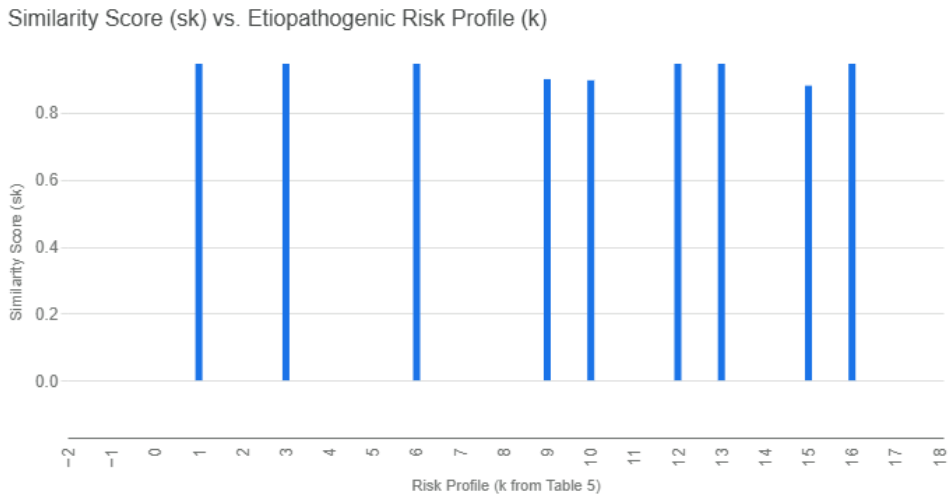

Figure 1. Degree of Correlation between Risk Factors and Phenotypic Expression for 16 Profiles.

Applying Equation 11 to calculate s_T :

$$s_T = \frac{\left(\Sigma_{k=1}^{16} s_k\right)}{16}$$

Addition $= 0.9533654 + 0 + 0.9473684 + 0 + 0 + 0.9590909 + 0 + 0 + 0.9007398 + 0.8968621 + 0 + 0.9805713 + 0.9473684 + 0 + 0.8809183 + 0.9495856$

Addition= 8.4158702

$$s_T = \frac{8.4158702}{16}$$

$$s_T = 0.5259918875$$

Rounding to the number of decimal places shown in the example:

$$s_T = 0.52599189$$

Classification:

According to Definition 9, two PSSs are considered "significantly similar" if:

$$S(F_P^z, G_P^z) \geq \frac{1}{2}$$

In our case:

$$s_T = 0.52599189 > 0.5$$

This is interpreted as the etiopathogenic risk profiles (the combination of the dimensions dim $_1$, dim $_2$, dim $_3$) showing a **positive and significant relationship** with the Phenotypic Expression dimension (dim $_4$) in the studied patient cohort.

4. Discussion

The result sT = 0.52599189 is of great importance. Due to it being greater than the threshold 0.5, it confirms the central hypothesis of the research endeavor: the etiopathogenic risk factors are positively correlated with the phenotypic expression of FLAP and largely under high uncertainty management by PSS model.

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Yet sT = 0.52599189 is only marginally greater than 0.5. This indicates significant but highly complex association. Indeterminacy (wherein plitogenic logic comes into play) reveals that these three risk factors do not account for all the variance in phenotypic expression. There are probably other, unmeasured factors or stochastic relations that the model renders as indeterminacy.

Furthermore, there are seven s_k values (for k = 2, 4, 5, 7, 8, 11, 14) that yield zero. This means that for these risk profiles (e.g., ε_2 = (RA, RMA, RMA, RMA)), no similarity existed in the aggregated data of the 28 patients from risk factors and phenotype. From a clinical perspective, this is tremendously important as it suggests risk profiles that do not have a direct correlation with phenotypic expression in this population. Thus, subgroups may have either different etiopathogenic efforts occurring or protective efforts.

5. Conclusion

The findings (sT > 0.5) confirmed that the Plitogenic Fuzzy Soft Sets (PSS) model is a valid and reliable framework for classifying FLAP patients according to their etiopathogenic risk profiles. The model demonstrated a positive and significant relationship between risk dimensions and phenotypic expression (EF), effectively addressing uncertainty and ambiguity in clinical data and overlapping risk factors. This approach extends traditional anatomical classifications toward etiopathogenic risk assessment, supporting early interventions and personalized genetic counseling. Furthermore, the study recommends strengthening clinical and public health practices based on the identified risk profiles. Behavioral and environmental factors should be closely monitored to optimize preventive measures, such as folic acid supplementation. The development of targeted intervention protocols for specific risk cohorts (e.g., ε_{12}) is encouraged, along with the enhancement of data collection systems to capture indeterminacy and contradictory information. The integration of plitogenic and neutrosophic logic (z = 3) will enable more comprehensive analyses in future research.

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