AN EXPERT SYSTEM MODEL IN PSYCHIATRY FOR CASE FORMULATION AND TREATMENT DECISION SUPPORT

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Abstract: Whilst case formulation is a critical task in psychiatry, it is an unexplored area in the field of medical expert systems development, which has mostly focused on the diagnostic inference. Case formulation plays a more important role in planning, and individualising treatments compared to categorical diagnoses. Nevertheless, case formulation is considered to be challenging task even for clinicians due to the highly subjective nature of the psychiatric knowledge, and lack of defined criteria, which are available for diagnoses. Lack of conceptual model, which captures the depth and the complexity of the clinical knowledge and reasoning demonstrated by expert clinicians, is considered to be a one of the root causes of failures in previous approaches. Whilst the authors have described a conceptual model for diagnostic consultation in a separate paper, this paper describes the conceptual model for case formulation and treatment decision support, thus laying down a domain-specific theoretical foundation required for successful implementation of expert systems in psychiatry. The knowledgebase has been conceptualised as a hierarchically organised set of entities spanning the domains of diagnostic knowledge, etiological knowledge and treatment knowledge, through which an iterative inference is made using the logical inferences of abduction, deduction and induction.

1 INTRODUCTION

Case formulation is considered to be a crucial task in psychiatric assessments. It requires high level skills and knowledge, and is typically a key aspect of assessment in specialist examinations in psychiatry. When a patient is being assessed, case formulation is important because it provides the core framework for cohesive integration of the clinical knowledge, and directs clinicians towards the most appropriate treatment(s). Poorly constructed case formulation may result in poorly focussed consideration of the patient's main clinical issues, perhaps leading to sub-optimal or even inappropriate treatment decisions, which may adversely affect the patient. Whilst case formulation is such an important task, it is often considered to be too challenging, particularly for junior clinicians (McDermott et al., 1996), (Mellsop and Banzato, 2006). As a foundation for this research work, the authors have introduced a systematic method for developing psychiatric case formulation for clinicians (Fernando et al., 2011).

Because they are two parallel processes that utilise a common data set (i.e. clinical symptoms and the patient history) in psychiatric assessment, case formulation and making diagnoses are closely related. Whilst diagnoses are categorical and generic, case formulation provides a conceptualisation to closely understand the unique circumstances of the individual. Therefore, case formulation is extremely important in developing an individualised treatment plan. Nevertheless, case formulation is an unexplored area in the field of expert systems development, and the small number of psychiatry expert systems (e.g. DUNE (Hardt and MacFadden, 1987)) described in the literature mainly address diagnostic consultations. The authors have already introduced a conceptual model for diagnostic consultation in psychiatry (Fernando et al., 2011). This paper expands on the conceptual model to encompass case formulation and treatment decisions, thereby laying down a complete

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theoretical framework for building expert systems in psychiatry, and for future research.

We believe medical expert system development in general faces a number of challenges in relation to the domains of conceptual modelling, implementation, and social and organisational aspects. The main problems of the previous approaches(e.g. INTERNIS-1/ CADUCEUS (Wolfram, 1995); (Miller, 1984); CADIAG-1 and CADIAG-2 (Adlassnig and Kolarzs, 1986); Parsimonious Covering Theory (Reggia and Peng, 1987); A Process Model of Diagnostic Reasoning (Stausberg and Person, 1999) to development of medical expert systems include: failure to develop conceptual models that capture the depth of the domain; difficulties in developing a sufficiently large knowledgebase; and failure to take into consideration the social and organisational issues related to operational aspects of the implemented system. The authors have discussed these aspects in a separate paper (including the limitations of the previous approaches), and have proposed a development framework in order to overcome these challenges (Fernando et al., 2011). The very first step towards developing a successful medical expert system is developing a conceptual model that captures the depth and the complexity of clinical reasoning in specialised medical domains. This paper and the previous one attempt to achieve this first step, specifically in the field of psychiatry.

2 KNOWLEDGEBASE MODEL

The key to successful clinical inference is the structure of the knowledgebase. Whilst there are approaches in which the knowledgebase is independent from the inference process (e.g. CLASSIKA (Gappa et al., 1993), PROTÉGÉ (Tu et al., 1995), such approaches are deemed unsuitable for a highly specialised knowledge domain such as psychiatry, in which the inference mechanism is dependent on the knowledgebase structure from the clinician's perspective.

The knowledgebase encompasses three domains: diagnostic knowledge; etiological knowledge; and the treatment knowledge, which may be organized as a hierarchy as described in Figure-1. The diagnostic domain of the knowledgebase consists of layers representing respectively individual symptoms, and clinical phenomena, in which symptoms combine to form unique clinical phenomena. The etiological domain of the knowledgebase consists of layers representing respectively model concepts, and explanatory models, which can be derived from a number of etiological theories in psychiatry including ego-psychology (Freud 1923); self-psychology (Kohut, 2009); object-relations theory (Ogden, 1983); attachment theory (Bowlby, 1969); cognitive schema therapy model (Young et al., 2003); and Interpersonal Therapy Model (Weissman et al., 2000). Each explanatory model consists of a unique combination of model concepts. Each clinical phenomenon is related to one or more model concepts, thus bridging the diagnostic domain and the etiological domain of the knowledgebase. The treatment domain consists of layers representing respectively treatment components, and individual treatments. Each treatment comprises a unique combination of treatment components. Figure-2 explains the knowledgebase model using an example, in which the two symptoms "low self-confidence" and "oversensitivity to criticism" along with several other symptoms form the clinical phenomenon "Low self-esteem". Next, this clinical phenomenon is related to the model concept, "Cognitive schema of defective self" in the etiological knowledge domain. One explanatory model is shown in the next layer of the etiological knowledgebase, and it is made up of three model concepts: "Predisposing events", "Cognitive schema of defective self" and "Precipitating events". This explanatory model is related to the treatment component "Cognitive Re-structuring" in the treatment knowledge domain, which happens to be a part of the treatment "Cognitive Behaviour Therapy". The clinical basis of this structure of the knowledgebase is not within the scope of this paper and is covered elsewhere (Fernando et al., 2011).

Clinical phenomena are made up of a constellation of symptoms, and arguably play a more critical role in clinical reasoning in psychiatry compared to other branches of medicine. They are directly related to phenomenological concepts in psychiatry, and can be considered as core clinical features or recurrent themes in clinical scenarios. Diagnostic inference based on clinical phenomena is considered to have more reliability and validity compared to that based on symptoms, since each clinical phenomenon is a unique constellation of a number of clinical symptoms.

The main components of the diagnostic knowledgebase and their relations are defined as follows.

$Symp = \{S_1, S_2, \dots, S_m\}$ is the set of all symptoms.

$Phen = \{P_1, P_2, \dots, P_n\}$ is the set of all clinical phenomena.

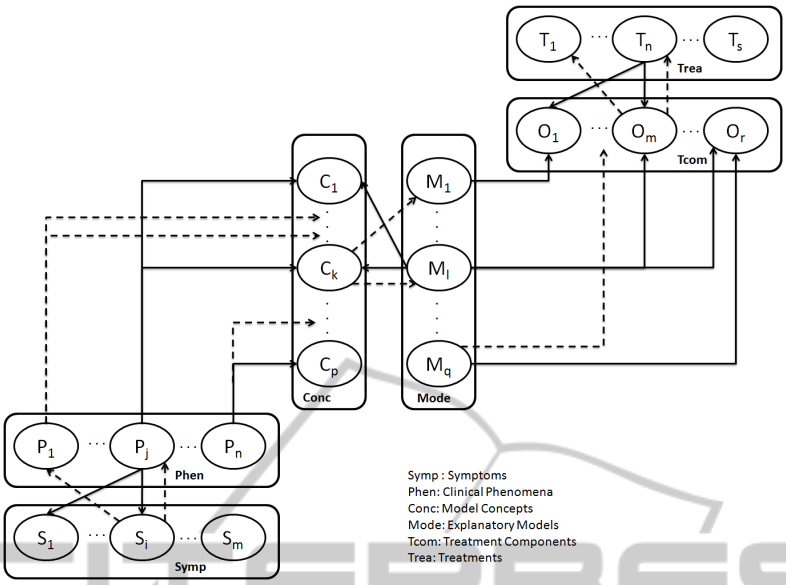

Figure 1: Hierarchical model of the three clusters of knowledge.

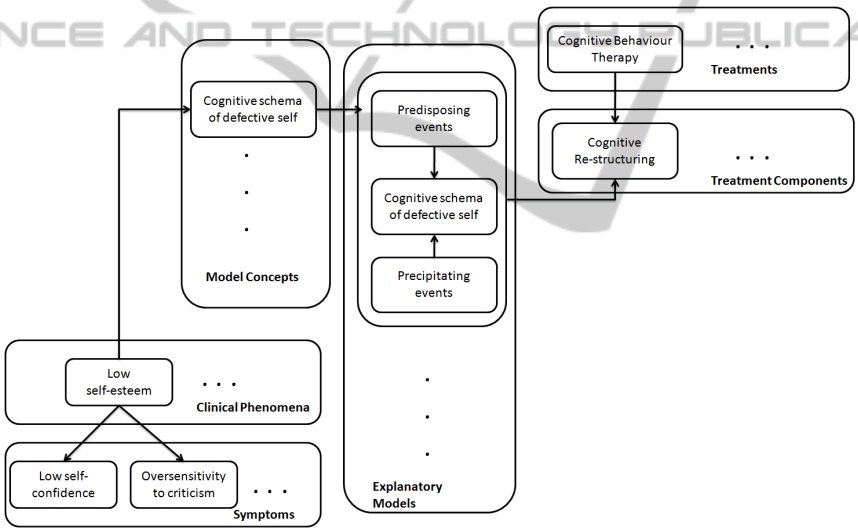

Figure 2: An example using the knowledgebase model.

$R_{PS} \subseteq Phen \times Symp = \langle P_j | S_i \rangle$ *where* $P_j \in Phen, S_i \in Symp$.

$Symp(P_j) = \{S_i | \langle P_j | S_i \rangle \in R_{PS}\} \quad \forall P_j \in Phen$

is the set of all symptoms related to a clinical phenomenon P_j.

$\mu_q : Symp \rightarrow [0,1]$ is a function where $\mu_q(S_i)$ indicates the degree of severity of the symptom, S_i on a scale from 0-1(i.e. $\mu_q(S_i) = 0 \implies$ the severity of the symptom S_i is minimum, whereas$\mu_q(S_i) = 1 \implies$ the severity of the symptom S_i is maximum).

$\mu_c : Phen \rightarrow [0,1]$ is a function where $\mu_c(P_i)$ indicates the degree of confirmation of the clinical phenomenon P_i in a scale from 0-1(i.e. $\mu_c(P_i) = 0 \implies$ the clinical phenomenon P_i is least likely to be confirmed, whereas$\mu_q(P_i) = 1 \implies$ the clinical phenomenon P_i is most likely to be confirmed).

$C_{ji} : [0,1] \rightarrow [0,1]$, $with\ \mu_c(P_j) = C_{ji}\left(\mu_q(S_i)\right), \langle P_j | S_i \rangle \in R_{PS}$ is a function which determines the degree of confirmation of the clinical phenomenon, P_j based on the severity of the symptom, S_i.

$$\mu_q(S_1) \wedge \mu_q(S_2) \wedge \ldots \wedge \mu_q(S_n) \implies \mu_c(P_j)$$

$$= \bigwedge_{i=1}^{n} \mu_q(S_i) \in Symp(P_j) \Rightarrow \mu_c(P_j)$$

is a rule that calculate the degree of confirmation of the clinical phenomenon, P_j based on the collective severities of the related symptoms, $S_1, S_2, \ldots, S_n$ using the following formula.

$$\mu_c(P_j) = \frac{1}{n}\sum_{i=1}^{n} C_{ji}\left(\mu_q(S_i)\right)$$

Case formulation is mainly constructed using the etiological knowledge, which bridges the diagnostic knowledge and treatment knowledge. Whilst there are operationalised and defined diagnostic criteria, which can be used during diagnostic reasoning, such explicit rules are non-existing for psychiatric case formulation. On the other hand, case formulation is more complex since there can be many alternative case formulations derived from different theoretical orientations(as described above) causing ambiguity. As a solution the authors have introduced an approach to systematically organise this knowledge and derive patterns using templates(Fernando et al., 2011).

The main components and their relations to the aetiological knowledgebase and the diagnostic knowledgebase can be defined as follows.

$Conc = \{C_1, C_2, \ldots, C_k, \ldots, C_p\}$ is the set of all model concepts and explanatory models.

$Mode = \{M_1, M_2, \ldots, M_k, \ldots, M_q\}$ is the set of all explanatory models.

$R_{MC} \subseteq Mode \times Conc = \langle M_j | C_i \rangle \; where \; M_j \in Mode, C_i \in Conc$ is the relation between the above two sets.

$Conc(M_j) = \{C_i | \langle M_j | C_i \rangle \in R_{MC}\} \; \forall M_j \in Mode$ is the set of all model concepts associated with a given explanatory model, M_j.

$R_{PC} \subseteq Phen \times Conc = \langle P_j | C_i \rangle \; where \; P_j \in Phen, C_i \in Conc$ is the relation between the respective sets of clinical phenomena and model concepts.

$Conc(P_j) = \{C_i | \langle P_j | C_i \rangle \in R_{PC}\} \; \forall P_j \in Phene$ is the set of all model concepts associated with a given clinical phenomenon, P_j.

$\mu_c(C_i) = \mu_c(P_j), \forall C_i \in Conc(P_j)$ defines that the degree of confirmation of any model concept, C_i associated with the clinical phenomenon P_j, is the same as the severity of P_j.

$$(\mu_c(C_1) > \theta_1) \wedge (\mu_c(C_2) > \theta_2) \wedge \ldots \wedge (\mu_c(C_n) > \theta_n) \Rightarrow \mu_s(M_j)$$

$$= \bigwedge_{i=1}^{n} (\mu_c(C_i) > \theta_i) \Rightarrow \mu_s(M_j), C_i \in Conc(M_j)$$

is a rule to determine the strength(i.e. explanatory power) of a given explanatory model, M_j using each of its model concepts, C_i of which the degree of confirmation should be above a threshold value, θ_i for the explanatory model to be substantiated. Given that this rule is satisfied, the strength of the model can be calculated as,

$$\mu_s(M_j) = \sum_{i=1}^{n} \mu_c(C_i)$$

$f_M: Conc \rightarrow Mode, \; where \; M_i = f_M(C_i)$ is a function that maps any given model concept C_i to its corresponding model concept, M_i.

Psychiatric treatments include pharmacological and physical interventions, psychological interventions, and social interventions. Each treatment intervention can be conceptualised as having several treatment components. Whilst a single treatment intervention is more general in relation to a particular psychiatric diagnosis and the patient, its components are more specific (i.e. some components are more relevant and applicable than others). One main advantage of this conceptualisation is that it enables the clinician to individualise treatment. Each treatment intervention is also associated with a set of favourable factors, unfavourable factors, and contra indications, which have to be evaluated against each patient's circumstances. For example, if a treatment intervention is causing weight gain as a side effect, then it is considered to be an unfavourable factor for a patient who is already obese; a side effect of sedation is considered to be a favourable factor for a patient who is having sleep difficulties. On the other hand, contra indications imply that the treatment intervention should not be prescribed for the patient, who has a condition that contra indicates the intervention (e.g. Electroconvulsive therapy is contra indicated for a patient with elevated intracranial pressure).

The components of the treatment knowledge, their properties, and relations with the etiological knowledge can be defined as follows.

$Tcom = \{O_1, O_2, \ldots, O_k, \ldots, O_r\}$ is the set of all treatment components.

$Trea = \{T_1, T_2, \ldots, T_k, \ldots, T_s\}$ is the set of all available treatments.

$R_{TO} \subseteq Trea \times Tcom = \langle T_j | O_i \rangle \; where \; T_j \in Trea, O_i \in Tcom$ is the relation between the above sets of treatments and treatment components.

$Tcom(T_j) = \{O_i | \langle T_j | O_i \rangle \in R_{TO}\} \forall T_j \in Trea$ is the set of all treatment components associated with a given treatment, T_j.

$R_{MO} \subseteq Mode \times Tcom = \langle M_j | O_i \rangle \; where \; M_j \in Mode, O_i \in Tcom$ is the relation between the two sets, explanatory models and treatment components.

$Tcom(M_j) = \{O_i | \langle M_j | O_i \rangle \in R_{MO}\} \forall M_j \in Mode$ is the set of all treatment components associated with a given explanatory model, M_j.

$\mu_e: R_{MO} \rightarrow [0,1]$ is a function where $\mu_e(\langle M_j | O_i \rangle)$ indicates the effect size of the treatment component, O_i for the explanatory model, M_j, on a scale from 0-1(i.e. $\mu_e(O_i) = 0 \implies$ the effect size of O_i is minimum, whereas $\mu_e(O_i) = 1 \implies$ the effect size of O_i is maximum).

$\langle P_j | C_i \rangle \; with \; \mu_c(P_j) > \sigma_i \implies \langle M_i | O_k \rangle \; with \; \mu_e(\langle M_i | O_k \rangle)$ is a rule that indicates the effect size of the treatment component O_k for the clinical phenomenon P_j , which has a degree of confirmation above the threshold value, σ_i , and is associated with the explanatory model M_i via the model concept, C_i.

$Favo(T_j) = \{F_1, F_2, \dots, F_k\}$ is the set of favourable factors associated with treatment, T_j.

$Unfa(T_j) = \{U_1, U_2, \dots, U_m\}$ is the set of unfavourable factors associated with treatment, T_j.

$Cont(T_j) = \{N_1, N_2, \dots, N_n\}$ is the set of contraindications for treatment, T_j.

$\mu_q: \{Favo(T_j) \cup Unfa(T_j)\} \rightarrow [0,1]$ is a function that quantifies the degree of the favourable factors, and of the unfavourable factors.

The favourable factors, unfavourable factors and the contraindications can be expressed using the following generic form of inference rule.

$\mu_q(F_1) \wedge \mu_q(F_2) \wedge \dots \wedge \mu_q(F_k) \implies \mu_f(T_j)$ where,

$$\mu_f(T_j) = \sum_{i=1}^{k} F_i \, \varepsilon_i$$

(ε_i is the weight assigned to the favourable factor, F_i according to its importance).

Similarly,

$\mu_q(U_1) \wedge \mu_q(U_2) \wedge \dots \wedge \mu_q(U_m) \implies \mu_u(T_j)$ where,

$$\mu_u(T_j) = \sum_{i=1}^{m} U_i \, \varphi_i$$

(φ_i is the weight assigned to the unfavourable factor, U_i according to its significance).

On the other hand, contraindications do not require quantification, and can be expressed in the following form of generic inference rule.

$N_1 \vee N_2 \vee \dots \vee N_n \implies \neg T_j$,which indicates that if any of the contraindications are present, the treatment, T_j should not be prescribed.

3 INFERENCE MODEL

As in the case of diagnostic consultation that has been covered elsewhere (Fernando, Henskens et al. 2011), we adopted the ST-model (Ramoni, Stefanelli et al. 1992), which provides a sound framework based on the logical inferences of abduction, deduction and induction described by Charles Peirce (Peirce 1878). There are other reasoning strategies described in the literature (e.g. Hypothetico-deductive reasoning (Elstein et al., 1978); Pattern recognition and categorisation (Norman et al., 1992); Inductive and Scheme-inductive reasoning (Mandin et al., 1997); Forward and Backward reasoning (Hunt, 1989), (Patel and Groen, 1986)), but they are not as comprehensive as the ST- Model. The clinical inference involves an iterative process of three stages: abstraction; abduction; and deduction, leading to the induction stage.

3.1 Abstraction

Patients report their symptoms and disclose their history using their own terminology and language, whereas the components of the knowledgebase are specific concepts defined in the clinician's mind. Abstraction involves the process of substantiating these concepts (i.e. symptoms, clinical phenomena, model concepts, models) by mapping what patients report into them. For example, a patient might report "having a dark cloud over me", which will turn out to be abstracted as the symptom "depressed mood". Abstraction also involves determining the severity of each symptom, S_i and determining the degree of confirmation of each clinical phenomena, P_i based on the functional relationship, C_{ji}, which is a mathematical function approximated using the expert clinical judgement (an example is given in Figure-3).

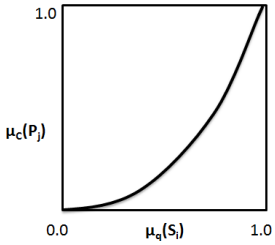

Figure 3: An example of the functional relationship between $\mu_q(S_i)$ and $\mu_c(P_j)$.

3.2 Abduction

After a symptom, S_i is substantiated via abstraction, abduction involves generating hypotheses, which indicates the likely clinical phenomena associated with S_i. Next, once a clinical phenomenon, P_j is substantiated (which also involves deduction and induction as described in following sections), abduction involves hypothesising the likely explanatory models. Similarly, once an explanatory model, M_k is substantiated, abduction involves hypothesising the possible treatment components indicated for M_k.

For example, consider the following two inference rules, in which S_i are included in the antecedent.

$$\mu_q(S_i) \wedge \mu_q(S_1) \wedge \mu_q(S_2) \implies \mu_c(P_j)$$
$$\mu_q(S_i) \wedge \mu_q(S_3) \wedge \mu_q(S_4) \implies \mu_c(P_k)$$

Once the degree of severity of S_i is determined, abduction involves hypothesising the clinical phenomena P_j and P_k, which, then, requires deductive inference as explained in the next section. Once, $\mu_c(P_j)$ and $\mu_c(P_k)$ are determined, abduction will infer the related model concepts, explanatory models, treatment components, and treatments in a similar manner. The direction of the abduction inference is bottom-up as indicated by the broken lines in Figure-1.

Abduction may involves generating a very large number of hypotheses, which may leads to an unacceptably lengthy inference cycle, impacting on efficiency. Strategies including prioritising the hypotheses, and using pattern recognition, and exclusion and inclusion criteria to narrow down the range of hypotheses have been discussed elsewhere(Fernando, Henskens et al. 2011).

3.3 Deduction

For each hypothesis generated during abduction, deduction involves exploring further information with the aim of confirming or rejecting the hypothesis. For example, as described under the above section, consider that the clinical phenomenon P_j is hypothesised via abduction based on the symptom S_i. Next, deduction involves eliciting symptoms S_1 and S_2 via abstraction, inferring $\mu_q(S_1)$ and $\mu_q(S_2)$, and finally, calculating $\mu_c(P_j)$ as follows.

$\mu_c(P_j) = \frac{1}{3}\left(C_{ji}(\mu_q(S_i)) + C_{j1}(\mu_q(S_1)) + C_{j2}(\mu_q(S_2))\right)$ where C_{ji} is the function, that describes the relationship between the severity of the symptom S_i denoted by $\mu_q(S_i)$ and the degree of the confirmation of the clinical phenomenon, P_j denoted by $\mu_c(P_j)$. Similarly C_{j1} is the function, that describes the relationship between $\mu_q(S_1)$ and $\mu_c(P_j)$; C_{j2} is the function, that describes the relationship between $\mu_q(S_2)$ and $\mu_c(P_j)$.

Similarly, for each explanatory model hypothesised, deduction involves exploring the rest of the model concepts, and therefore related clinical phenomena included in similar inference rules.

In relation to the process of making treatment decisions, deduction involves exploring contraindications, and favourable factors associated with each hypothesised treatment component, and treatment. For example, consider the following inference rules for favourable factors, unfavourable factors, and contraindications for the treatment, T_j which has been hypothesised.

$$F_1 \wedge F_2 \wedge \ldots \wedge F_k \implies T_j$$
$$U_1 \wedge U_2 \wedge \ldots \wedge U_m \implies T_j$$
$$N_1 \vee N_2 \vee \ldots \vee N_n \implies \neg T_j$$

Deduction involves exploring the presence of any of the favourable factors, $F_1, F_2, \ldots, F_k$; unfavourable factors $U_1, U_2, \ldots, U_m$; and the contraindications $N_1, N_2, \ldots, N_n$.

The direction of the deductive inference is the reverse of the abductive inference (i.e. top-down) as indicated by solid lines in Figure-1.

3.4 Induction

Inductive inference ends the iterative inference cycle, and involves accepting or rejecting the generated hypotheses using the information gathered during the previous stages, by matching them with the inference rules. For example, given the inference rule involving model concepts, $(\mu_c(C_1) > \theta_1) \wedge (\mu_c(C_2) > \theta_2) \wedge (\mu_c(C_n) > \theta_n) \implies \mu_s(M_j)$, and $\mu_c(C_1) > \theta_1$, $\mu_c(C_2) > \theta_2$ and $\mu_c(C_n) > \theta_n$

then the explanatory model M_j would be confirmed with a strength of $\mu_s(M_j)$.

Deciding the best treatment, involves calculating the effect size and then the suitability of each treatment using the following formula.

$$\mu_e(T_j) = \frac{1}{n}\sum_{i=1}^{n} Max\left(\mu_e(\langle M_k|O_i\rangle)\right),\ \forall O_i \in Tcom(T_j),\ \forall O_i \in Tcom(M_k),$$

which calculates the effect size of the treatment, T_j by taking the average value of the total sum of the maximum effect size related to each pair of its treatment component and explanatory model.

For example, consider the patient's clinical phenomena are explained by three explanatory models, M_1, M_2 and M_3, each of which is paired with three different treatment components O_1, O_2 and O_3 as follows:

$\langle M_1|O_2\rangle, \langle M_2|O_1\rangle, \langle M_3|O_1\rangle, \langle M_1|O_3\rangle, \langle M_2|O_2\rangle$ and $\langle M_3|O_2\rangle$. Suppose that the effect-size of these tuples are respectively $\mu_e(\langle M_1|O_2\rangle) = 0.4$; $\mu_e(\langle M_2|O_1\rangle) = 0.6$; $\mu_e(\langle M_3|O_1\rangle) = 0.8$; $\mu_e(\langle M_1|O_3\rangle) = 0.2$; $\mu_e(\langle M_2|O_2\rangle) = 0.3$; $\mu_e(\langle M_3|O_2\rangle) = 0.7$.

Now, consider that there are two treatments, T_1 andT_2 which are paired with the treatment components as follows:

$\langle T_1|O_1\rangle, \langle T_1|O_2\rangle$ and $\langle T_2|O_1\rangle, \langle T_2|O_3\rangle$ Determining whether treatment, T_1 or T_2 has the higher effect size involves the following calculations:

$$\mu_e(T_1) = \frac{1}{2}\big(Max(\mu_e(\langle M_2|O_1\rangle), \mu_e(\langle M_3|O_1\rangle)) + Max(\mu_e(\mu_e(\langle M_1|O_2\rangle), \langle M_2|O_2\rangle), \mu_e(\langle M_3|O_2\rangle))\big)$$
$$= \frac{1}{2}\big(Max(0.6,0.8) + Max(0.4,0.3,0.7)\big)$$
$$= \frac{1}{2}(0.8 + 0.7) = 0.75$$

$$\mu_e(T_2) = \frac{1}{2}(Max(\mu_e(\langle M_2|O_1\rangle), \mu_e(\langle M_3|O_1\rangle)) + Max(\mu_e(\langle M_1|O_3\rangle)))$$
$$= \frac{1}{2}\big(Max(0.6,0.8) + Max(0.2)\big)$$
$$= \frac{1}{2}(0.8 + 0.2) = 0.5$$

Therefore T_1 has the higher effect-size.

Next, the overall suitability of a given treatment, T_j is calculated based on the effect size and the cumulative effect of favourable and unfavourable factors, using the formula,

$$\mu_s(T_j) = \mu_e(T_j)\left(\sum_{i=1}^{n} F_i\,\varepsilon_i - \sum_{i=1}^{m} U_i\,\varphi_i\right)$$

4 CONCLUSIONS

Medical expert systems have not progressed much after an initial golden era several decades ago. The authors have identified a number of reasons related to developing conceptual and computational models, their implementations and social issues. The root cause of the failure, however, is related to the difficulty of capturing the depth and the complexity of broader clinical reasoning (involving all three aspects of diagnostic assessment, etiological formulation and treatment decisions) exhibited by expert clinicians. A further problem involves that of engrossing the expert's reasoning in a conceptual model, when knowledge engineers do not have the necessary medical background. Additionally, generic medical expert system models are unsuitable, since there are significant differences in relation to the nature of the domain knowledge and the inference strategies used in different medical specialties. Furthermore, the inference mechanism is dependent on the structure of the knowledgebase.

As a new approach, the authors have proposed a conceptual model for developing a domain-specific expert system in psychiatry. This paper addresses etiological reasoning, which involves case formulation, and treatment decisions in psychiatry; the authors previously addressed the issue of diagnostic reasoning in a separate and complementary paper (Fernando et al., 2011). The pair of papers thus completely cover the broader aspects of clinical reasoning in psychiatry. Importantly, whilst the crucial role of case formulation in psychiatry has been previously recognised, we have for the first time modeled case formulation in a way that can be implemented in psychiatry-specific expert systems. Whilst this conceptual model will undoubtedly be subject to future revision and refinement, it completes the first step required for developing successful expert system applications. Finally, it is expected that the theoretical foundation described here will provide insight to development of expert systems in other medical specialties.

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