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與健康有關的生命品質的因果本體論和因果複雜性

Causal Complexity and Causal Ontology of Health-Related Quality of Life Model

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Thanks to my professor Dumbledore, who sacrificed himself for the victory.

與健康有關的生命品質的因果本體論和因果複雜性

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摘　　要

以病患爲中心的照護 (patient-centered care, PCC) 是一個重視病人喜好，需要與自治的照護取向。評估 PCC 的實踐是評估醫療照護的一部分。而且，臨床研究的結果可以爲評估 PCC 的實踐提供重要資訊。在臨床研究中，與健康有關的生活品質 (health-related quality of life, HRQL) 的理論模型爲臨床研究提供了概念的工具箱，並引導臨床研究中的假設生成。Wilson and Cleary (1995) 發展了最爲廣泛使用的 HRQL 理論模型。在 Wilson and Cleary 的模型中的因果本體論假設會影響哪一種因果假設將在研究中被生成。我將論證 HRQL 的臨床研究被 Wilson and Cleary 的模型灌輸了一種因果偏誤: 從生物醫學因素到非生物醫學因素的因果假設很常被生成，但非生物醫學因素到生物醫學因素的因果假設卻很少被生成。這樣的因果偏誤造成了病人的組成部分之間的互相依賴與互動被忽略，且這個後果是實踐 PCC 的阻礙。接著，我將會提供一個修正版本的 HRQL 理論模型，這個修正方案避免了上述的因果偏誤，並參考了與 HRQL 臨床研究有關的重要洞見。透過本文的工作，我爲透過分析因果本體論的假設來使醫療照護的實作進步騰出了空間。

關鍵詞: 與健康有關的生活品質，生活品質，以病人爲中心的照護，因果本體論，因果複雜性。

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Abstract

Patient-centered care (PCC) is an approach to healthcare that values patients' preference, need, and autonomy. The estimation of healthcare partly depends on how well PCC is implemented. In addition, the result of clinical research can inform the assessment of the implementation of PCC. In clinical research, health-related quality of life (HRQL) theoretical models offer a conceptual toolbox that informs clinical research and guides the hypotheses generation. Wilson and Cleary (1995) developed the most widely used HRQL theoretical model (Bakas et al., 2012). Ontological assumptions about causation in Wilson and Cleary's model will influence which kind of hypotheses will be generated. I will argue that Wilson and Cleary's model instilled a kind of causal bias into hypothesis generation in clinical research on HRQL. Causation from biomedical factors to non-biomedical factors is frequently hypothesized while causation from non-biomedical factors to biomedical factors is rarely hypothesized. It leads to that the interdependence and interaction between constituent parts of patients are ignored, which is an obstacle to the implementation of PCC. In addition, I will propose a revised HRQL theoretical model which avoids the causal bias brought by Wilson and Cleary's model. By doing so, I leave room for the improvement of the practice of healthcare by analyzing the ontological assumptions about causation.

Keyword: Health-related quality of life, Patient-centered care, causal ontology, causal complexity

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1. Introduction

Patient-centered care (PCC) promotes the kind of healthcare that values patients' rights, perspectives, and autonomy. The result of clinical research on health-related quality of life (HRQL) can help assess how well the implementation of PCC is. HRQL is a construct that consists of different dimensions of patients' health conditions, such as biomedical factors, functional status, general health perception, and overall quality of life (McClimans, 2019; Wilson and Cleary, 1995). Clinical practitioners usually employ HRQL theoretical models to generate hypotheses about how different conditions of patients influence each other. Wilson and Cleary (1995) developed the most widely-used theoretical model that informed the clinical research on HRQL (Bakas et al., 2012). In this paper I will use 'the W&C model' to refer to Wilson and Cleary's model.

In this paper, I will point out that the W&C model implicitly instills a causal bias into the current HRQL measuring practice, even though they do not explicitly endorse any causal ontology in their model (1995, p. 60). Causal ontology refers to those presumptions contained in a model, which involve the commitment of causation, e.g., only the biomedical factors can have causal power. Based on my literature analysis, most of the HRQL research guided by W&C model has the same type of causal hypotheses, i.e., from biomedical factors to non-biomedical factors. Causal hypotheses regarding how the changes in non-biomedical factors cause the changes in biomedical factors are rarely investigated. This causal bias is an obstacle for implementing PCC because it implicitly directs researchers' attention away from how patients' values, preferences, and overall quality of life can causally affect their HRQL.

To rectify this implicit causal bias that impedes PCC implementation, I will propose a way to strengthen the causal ontology of W&C model. I will employ Rocca and Anjum's (2020) notion of causal complexity to modify Wilson and Cleary's model. Rocca and

Anjum (2020) thought the genuine complexity should embrace that variables from different dimensions of a patient can cause each other or co-cause an illness. I propose to change how Wilson and Cleary present causal connections in their diagram to represent their theoretical model. I retain all variables except non-medical factors while deleting the arrow and lines within W&C model. Instead, I use dotted lines to link all variables with each other in order to avoid the causal bias that W&C model did and leave room for the influential role of emergent approach. Since the emergent approach will be focused on in my proposal, the causal hypotheses regarding how changes in non-biomedical factors cause changes in biomedical factors will receive much more attention. In other words, my proposed changes will provide clear guidance and motivation for clinical researchers to investigate how patients' values, preferences, and overall quality of life can causally affect their HRQL.

The structure of this paper is as follows: Sect. 2 will explain what HRQL and PCC are. This is necessary because I will employ the connection between HRQL and PCC to construct the premises of my argument. The connection will be revealed as an account of how HRQL theoretical models serve healthcare that promotes PCC. This account implies that the causal ontology of HRQL theoretical models and of PCC should be at least consistent. Sect. 3 analyzes the causal ontology of PCC and W&C model, which is the focused HRQL theoretical model in this paper. In this step, I argue that the causal ontology of W&C model is consistent with the causal ontology of PCC. However, although W&C model seems qualified to serve healthcare, I will point out that W&C model instills causal bias which excludes the generation of causal hypotheses that from non-biomedical factors to biomedical factors into clinical research. In Sect. 4, I will review some works relevant to the revision of the HRQL theoretical model. These works informed the modification of W&C model. At the end of this paper, I will also offer my proposal, which is a topological model, as a solution.

2. Health-Related Quality of Life and Patient-Centered Care

2.1 Health-Related Quality of Life and Patient-Centered Care

'Health-related quality of life' (HRQL) refers to aspects of quality of life which related to health status. In addition, 'quality of life' is understood in association with happiness or life satisfaction (Fayers and Machin, 2002; Alexandrova 2017). Overall quality of life may be affected by economic, political, and cultural factors, yet in the context of health-care, those factors related to health status are considered more (Wilson and Cleary, 1995). Thus, clinical researchers employ the concept of HRQL to exclude those aspects of quality of life that have no relevance to health status.

To understand more about what HRQL is, a example is helpful. The Karnofsky performance scale (KPS) was proposed in 1947 and is generally thought to be the first instrument that "broadened the assessment of patients beyond physiological and clinical examination" (Karnofsky and Burchenal, 1952; Fayers and Machin, 2002, p.7). Timmerman (2012) also discussed KPS. He stated the objectives of his work, "[t]o use the history of the Karnofsky Performance Scale as a case study illustrating the emergence of interest in the measurement and standardisation of quality of life…" (p.179). These works give us a rough sketch of HRQL through how KPS was used. KPS shed the light on that some health-related measurement tools could be used to assess not only the biomedical factors of the patients, such as blood pressure or life expectancy but also non-biomedical factors, such as the functional status of the patients, such as how well the patients can perform daily activities. In short, HRQL could be identified as those health-related factors assessed by

HRQL measurement tools (e.g., KPS) including biomedical and non-biomedical factors.

According to the Institute of Medicine (IOM, 2001), the definition of patient-centered care (PCC) is "providing care that is respectful of, and responsive to, individual patient preferences, needs and values, and ensuring that patient values guide all clinical decisions." (p.6). To give a more clear characterization of PCC, I have to talk about the emergence of the new medical ethics. The 'term new medical ethics' was discussed by Faden and Beauchamp (1986), and also invoked in McClimans (2021). Since the second half of the twentieth century, physicians were required to inform patients about their illnesses, and patients were empowered to make decisions about their illnesses (Faden and Beauchamp, 1986). The change in the ethical requirement showed the change in doctor-relationship: physicians can no longer be silent and dominate the decision on patients' illnesses. The spirit of the new medical ethics was embedded in the Patient's Bill of Rights, which was developed in 1973 by the American Hospital Association (McClimans, 2021; Faden and Beauchamp, 1986). Since we have seen that the spirit of the new medical ethics is embedded in a bill, which is a proposed law, it is reasonable to say that the new medical ethics is the consensus of healthcare. In light of the new medical ethics, qualified healthcare should be implemented in a way that values not only the longer length of life or the stable biomedical factors but also patients' rights, perspectives, and autonomy (McClimans, 2021). If we compare the definition of PCC and the characteristics of the new medical ethics, we can naturally find several similarities between them. They all agree that patients' needs and perspectives are valued and patients should be empowered to make decisions about their illnesses.

The sketch of HRQL and PCC above are enough to construct an account of how HRQL serve healthcare that promotes PCC, which will be discussed in the following subsection.

2.2 The Way HRQL Serve Healthcare as a Vehicle for PCC

I aim to improve the practice of healthcare that promotes PCC by analyzing the ontological assumptions about causation in HRQL theoretical model. To enable this ambition, I have to elaborate on the connection between HRQL and PCC. Otherwise, the analysis has no chance to contribute to the healthcare that promotes PCC. To arrange the connection is the necessary condition of my goal.

McClimans (2021) argued that HRQL "serve healthcare as vehicles for patient-centered care⋯" (p.2526). McClimans (2021) thought HRQL should be distinguished from the other concept of well-being since HRQL uniquely has a historical context with PCC, but the other concepts of well-being do not. She invoked the development of HRQL to elaborate on the special relationship between HRQL and PCC. PCC is an approach to healthcare that values patients' needs and perspectives. HRQL measurement tools did an alternatively good job of capturing patients' perspectives, therefore, HRQL measurement tools became popular with practitioners (McClimans, 2021). Because the other concepts of well-being and their measurement tools were not used particularly in the context of healthcare or in the context of individuals, they are not strongly connected to PCC as HRQL. McClimans concluded that HRQL measurement tools are vehicles for PCC (McClimans, 2021). In sum, since what PCC commit apart HRQL from the other measures derived from the other theories of well-being, a strong connection between PCC and HRQL has been established (McClimans, 2021). As vehicles for PCC, HRQL is investigated in clinical research in order to improve the implementation of PCC.

Notice that McClimans (2021) used 'HRQL' to refer to measures of HRQL, such as KPS and the Patient Generated Index (PGI). Is there connection between measures of HRQL, like KPS, and PCC? 'HRQL' can refers to different things that works together

in clinical research: a construct, measures of the construct, and models that describe the construct. I use the term 'HRQL', 'HRQL measurement tools' and 'HRQL theoretical models' to refer to them in the rest of this paper. HRQL measurement tools measure the change in the construct, and HRQL theoretical models give a framework to describe the construct. For example, Frank et al. (2004) generated hypotheses about the relationship between the different aspects of a patient's health status. He investigated whether the change of a factor of health status would influence the other. The distinction between the different aspects of a patient's health status was based on the W&C model. In this work, HRQL measurement tools such as The Short Form (36) Health Survey (SF-36) were used (Frank et al., 2004). The pathway is what follows: clinical research invokes HRQL theoretical models to generate hypotheses, then researchers pick appropriate HRQL measurement tools to test the hypotheses generated. The result of healthcare research can help practitioners assess how well healthcare that promotes PCC is delivered. In short, HRQL theoretical models inform the research, including hypothesis generation and selection of HRQL measurement tools. Research in healthcare interprets the outcomes of patients and guides practitioners to improve healthcare that promotes PCC. If so, then there is a connection not only between HRQL measurement tools and PCC but also between HRQL theoretical models and PCC. Thus the isssue on HRQL theoretical model is supposed to be related to the implementation of PCC as well.

McClimans (2021) elaborated on the relationship between PCC and HRQL meausrement tools, while I shift the focus to the relationship between PCC and HRQL theretical models. Both two are routes that potentially affect the practice of healthcare that promotes PCC. The route that improving healthcare by analyzing ontological assumptions about causation of HRQL theoretical models has its merit. A theoretical model contains several presumed philosophical assumptions, and so does the W&C model. If philosophical assumptions of the HRQL theoretical models and PCC are not consistent with each other, then it is not convincing to say that HRQL can serve healthcare as we expected.

I will give an example. Say, one ontological assumption of PCC is to treat patients as a whole, while one ontological assumption of an HRQL theoretical model, which I call M1, is to treat patients as the summation consisting of cells and organs. This ontological assumption is not consistent with what PCC commits. The researcher generates a hypothesis, which I call H1, based on M1. The other ontological assumption that the patients are individuals as a complex whole is committed in theoretical HRQL model, which I call M2. Researchers also generate a hypothesis, which I call H2, based on M2. When comparing these two cases, it makes more sense that the result of the research used M2 and tested H2 could be identified as at least potentially helpful in improving the healthcare that promotes PCC. Toward this, the ontological assumptions of HRQL theoretical models should be analyzed, since it heavily influences the hypothesis generation and the selection of HRQL measurement tools. If I can exclude the inappropriate ontological assumptions of HRQL theoretical models or rectify the ontological assumptions of HRQL theoretical models to more fit PCC, then there would likely to be a positive influence from the change from causal ontology to the change of practice of healthcare that promotes PCC.

At the end of this section, I'd like to clarify something. I will not deal with the issue regarding whether clinical practitioners should pursue PCC or not. My analysis aims at evaluating theoretical models that inform how practitioners generate hypotheses. In the next section, we will see a concrete case that HRQL theoretical heavily influence the hypothesis generation. Thus, analyzing the ontological assumptions about causation in HRQL theoretical models is a significant work to contribute to the healthcare that promotes PCC.

3. The Ontological Assumptions about Causation in PCC and the W&C model, and the Causal Bias.

In this section, I will analyze the ontological assumptions about causation in PCC and the W&C model, specifically whether they are consistent. I will show that, although the relevant causal assumptions are consistent, the W&C model instills a kind of causal bias in clinical researchers' ways of generating causal hypotheses, i.e., hypotheses about how the change in biomedical factors cause the change in non-biomedical factors. In addition, I argue the W&C model is responsible for the causal bias, which is an obstacle to the implementation of PCC.

3.1 The Ontological Assumptions about Causation in PCC

PCC centers patients when they develop and organize their care practice. But how do clinical practitioners understand the nature of patients in the context of measuring HRQL? There are many factors of patients that can influence their HRQL, the interrelationship of these factors makes the patients complex wholes. What does it mean to say that patients are complex whole? In the philosophy of science, there are at least two ways of construing complexity. One is the mereological approach and the other is the emergent approach (Rocca and Anjum, 2020). Under the mereological approach, patients as a biological complex whole are composed by underlying biological parts. Furthermore, a biological complex whole is nothing but the sum of its parts. Under the emergent approach, a complex whole is more than, or something else than the sum of its parts. I will give a couple of examples to illustrate them.

A car is a good metaphor to understand the mereological approach. Imagine a car and its engine, tires, windows, seats, etc. The engine had some problems, and the car was

sent to a repair shop. The technician broke the car down into several parts and examined the engine situation. After a repair process, the engine was identified as broken, and it was fixed and then put back into the car. In this case, we encountered a question about causation: What is the cause of the unfortunate event to the car? Under the mereological approach, the car is merely the sum of engine, tires, windows, seats, etc. This understanding of the complex whole gives a starting point to answer the question: To examine the constituent parts of the car separately and independently. So a kind of causal hypothesis is possible: the change in engine (the part) cause the change in the car (the whole). The causal power can be attributed to the broken engine without involving the other causes.

An example of the emergent approach is the environment where beavers live and interact with their surroundings (Rocca and Anjum, 2020). While beavers build a dam, which is an action to change the surroundings, the surroundings also change the beavers by natural selection. Supposed one day, the environment becomes easier to be in flood, what is the starting point to generate the causal hypothesis? It is hard to merely attribute the causal power to the numbers of the beavers declining, or other changes in the surroundings. Because the interaction between the surroundings and the beavers is complex and there is no way only the numbers of the beavers changed but the surroundings did not change. The environment is the result of the process that which the beavers and the surroundings influence each other. So, the causal power could be attributed to the beavers *in the context of the environment* but not the beavers themselves. If we don't consider anything about the surroundings and the environment as the complex whole incorporated the beavers, the beavers do not have the causal power to the flood. Thus, the starting point of generating the causal hypothesis to the environment that becomes easier being in flood is to consider the interaction between constituent parts. It is different from the mereological approach since there is no need to consider the interaction in the hypotheses generation of the mereological approach.

Now, we might ask, in PCC, patients are treated as a complex whole in terms of emergent approach, or in terms of mereological approach? I aim to argue that it is in terms of the emergent approach by examine a question: Do the constituent parts of patients be separately examined in healthcare that promotes PCC? No. Practitioners who approach PCC deliver healthcare that values the different needs and preferences of patients (Lusk, 2013; IOM, 2001). It reveals that the context of each patient is considered when the healthcare is delivered, which is indicated by the variation of preference and needs. What is needed in delivering healthcare with the mereological approach is to care for the constituent parts that are responsible for the suffering of the patients. But in the practice of healthcare that promotes PCC, it is not the case that consider merely the economic conditions or the biomedical conditions of a patient, for instance. These conditions interact with each other *in the context of the patient*, thus the preference and needs vary in different contexts of patients. The way with the mereological approach is not capable to deal with the variation of preferences and needs of patients (Lusk, 2013; Morgan and Yoder, 2012). Since the constituent parts of patients are not separately examined in the healthcare that promotes PCC, I conclude the emergent approach is endorsed within PCC.

I have argued that the healthcare that promotes PCC is delivered with the emergent approach. And there are two central ontological assumptions about causation that can be derived: 1) Parts of patients are interdependent, and 2) different dimensions of patients can co-cause the change in the patient as a whole. The term 'interdependent' refers to that causation in other directions must be met in a causal inquiry. Rocca and Anjum (2020) sketch two kinds of causality, which I use the term 'direction of causation' to characterize: bottom-up and top-down. A diagnosis that a headache is caused by hormonal fluctuations, is bottom-up causality. A diagnosis that a headache is caused by financial worries in times of economic recession is top-down causality. Both bottom-up and top-down indicate the concept of 'direction' I used. The concept of 'direction' can also be indicated in terms of biomedical factors to non-biomedical factors, for instance. Recall to 1) since the interac-

tion between parts of patients is considered when investigating what is responsible for the change in patients as wholes, only hypothesizing one direction of causation is unpleasant. Suppose that we are inquiring about the causation of the headache case. When we only hypothesize bottom-up causality, we assume the credit of causal power should give to hormonal fluctuations while whether financial worries have causal power in the inquiry is ignored. If so, the consideration will be mere whether hormonal fluctuations are responsible for the headache, without the need that considers the changes in any other factors (e.g., non-biomedical factors) that are responsible for the change in hormonal fluctuations or the headache, and so on. It is not allowed in light of the emergent approach, therefore, different directions of causation must be met in causal inquiry. In addition, if we want to examine the interaction between constituent parts of patients, then the possibility that different dimensions can co-cause the change in patients as a whole must be guaranteed. To refuse the possibility is to accept that there can be only one dimension of the patient to be the cause of the change in the patient as a whole. However, the 'context' includes the non-biomedical factors and biomedical factors which interdependent with each other. So there cannot be only one factor that has the causal power to the change in the patient as a whole.

3.2 The Ontological Assumptions about Causation in the W&C Model

I have elaborated on the ontological assumptions about causation in PCC. In Sect. 1, I have argued that the ontological assumptions about causation in the W&C model should be consistent with those assumed in PCC. In this subsection, I will offer an description of the W&C model, and I will argue that the ontological assumptions about causation in the W&C model are consistent with those in PCC.

Wilson and Cleary (1995) aimed to "integrate different types of patient outcome measures by linking the biomedical model and the quality of life model" (p.59). They offered a theoretical model of HRQL that distinguishes five levels of HRQL with arrows connected each of them (see Figure 3.1).

From level one to level five are the biological and physiological variables, symptom status, functional status, general health perceptions, and overall quality of life. These five levels of HRQL do not refer to HRQL measurement tools but refer to HRQL constructs. The arrows represent the "dominant causal associations" (p.60). For example, there is an arrow from biological and physiological variables to the status of the symptoms. The arrow represents the relationship that the change of the former causes the change of the latter. But Wilson and Cleary (1995) stated that "[t]he arrows in the Figure [i.e., Figure 3.1] do not imply that there are not reciprocal relationships. Neither does the absence of arrows between nonadjacent levels imply that there are not such relationships" (p.60). The relationship represented by the arrows is the dominant, but not the only one. The changes in the characteristics of the individual, the environment, and non-medical factors can also cause the change in HRQLs.

To analyze whether the ontological assumptions about causation in PCC and the W&C model are consistent, I will raised three questions. The first is whether the W&C model

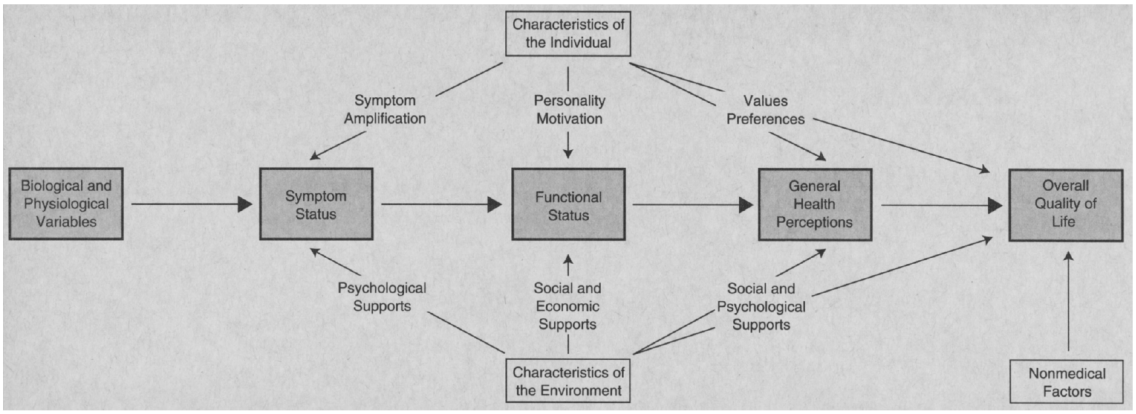

Figure 3.1: The W&C model. Reproduced from Wilson and Cleary (1995).

is a reductionistic model. Reductionism is an idea that all process and events must be the result of physical causes (Rocca and Anjum, 2020). In the description of the W&C model, the biological and physiological variables seem like always be the cause of the change in the other HRQL. If this is the case, then the W&C model is a reductionistic model. However, being a reductionistic model will be contradicted with the ontological assumption that parts of patients are interdependent since there is no way that changes in higher-level HRQLs cause the change in biological and physioilogical variables. I will argue the W&C model is not reductionistic model. The second is whether the W&C model contain only bottom-up causation. Although the reductionism can be rejected, it is not sufficient to leave rooms for the interdependence because it seems like only bottom-up causality is contained in the W&C model. I will argue there are causation in other directions in the W&C model, hence the interdependence is possible. The third is whether the two ontological assumptions about causation are endorsed in the W&C model. I aim to make sure the two ontological assumptions are committed in the W&C model. I will argue these assumptions are met in the W&C model according to the description of the W&C model.

Let's begin with the first question: Is the W&C model a reductionistic model? Wilson and Cleary (1995) stated that level one HRQL is "[t]he most fundamental determinants of health" (p.60). This is probably the main reason that there is only an arrow from biological and physiological variables to other HRQLs and no arrow from the other HRQLs to biological and physiological variables. This can be understood as indicating that only the change in biological and physiological variables can be the cause of the change in the other HRQLs. If so, then the W&C model is a reductionistic model because the change in biological and physiological variables are the physical cause and all changes in the other HRQLs are ultimately the results. However, there is an arrow from general health perception to the overall quality of life. The general health perception include non-physical components, and this is the counterexample that there is a non-physical cause in the W&C model. According to Wilson and Cleary (1995), "[t]wo salient characteristics of general

health perceptions are that they represent an integration of all the health concepts that we have previously discussed, as well as others such as mental health, and are by definition a subjective rating" (p.62). The non-physical component of it can be identified. Since there are non-physical components of level four HRQL and at least the causation from level four HRQL to level five HRQL is guaranteed by the W&C model, therefore, at least the possibility that the change of level five HRQL is caused by non-physical variables remains. I conclude that the W&C model is not a reductionistic model.

The second question is that: Whether the W&C model contain only bottom-up causation? If the answer is yes, then there is no top-down causality or causation in other directions in the W&C model. The ontological assumptions about causation in the W&C model are thus not consistent with those in PCC. Yet the description of arrows in the W&C model is vague. It leaves rooms to top-down causality and causation in other directions. I have quoted the description above, we have known that the arrows merely represent the dominant causal association, in other words, there are other possibilities on the causation between level one HRQL and the other HRQLs except the bottom-up causation. Since Wilson and Cleary (1995) did not deny these possibilities, it makes no sense to claim that there is only bottom-up causality in the W&C model. I will give the other reason. The rejection of top-down causality or causation in other directions in the W&C model is equivalent to say it is impossible that the lower level HRQL has changed while the higher level HRQL has not. However, there is counterexample in the W&C model. Here is the case of clinical research to support my claim. Wilson and Cleary (1995) said, "in a study of patients undergoing prostatectomy, it was found that among patients with 'severe' symptoms, 32% reported no day-to-day limitations because of their prostate condition, and 19% reported no worry about their health because of their prostate." I transform this quotation in terms of levels of HRQLs. 32% of patients undergoing prostatectomy reported that level two HRQL has changed while level three HRQL does not change, and there are 19% of them reported that level two HRQL has changed while level four HRQL does not change.

I conclude there is bottom-up causality or causation in other directions in the W&C model.

The third questions is that whether the two ontological assumptions about causation are endorsed in the W&C model. These assumptions are 1) parts of patients are interdependent, and 2) different dimensions of patients can co-cause the illness. 1) is satisfied by the description of the arrows and the case of patients undergoing prostatectomy. Since Wilson and Cleary (1995) did not exclude the possibilities that the change of high-level HRQL can cause the change of low-level HRQL and that there might be causation between two HRQLs without arrows linking each other, it makes sense to say parts of patients are interdependent. 2) is also included. There are arrows from the characteristics of the individual to functional status, from the characteristics of the environment to functional status, and from the symptom status to functional status. This guarantees that different dimensions of patients can be the cause of a change in HRQL in a patient. Therefore, I conclude that the W&C model is not a reducitonistic model, it contians not only bottom-up causality, and it incorporates ontological assumptions about causation which are contained in PCC.

I have argued that the two ontological assumptions about causation are met in the W&C model. But I want to point out there is a disadvantage of the W&C model, which will be discussed in the next subsection.

3.3 The Bias Instilled by the W&C Model into Clinical Research of HRQL

I want to point out there is a causal bias instilled by the W&C model to clinical researchers. For clinical researchers, it is natural to use the W&C model to generate bottom-up causal hypotheses. But it is relatively hard to use the W&C model to generate top-down causal hypotheses since there is less information regarding causation from non-biomedical

factors to biomedical factors. Therefore, the result of clinical research give much information about bottom-up causality but little about top-down causality or causation in other directions. If this is the case, there is *in fact* no difference between that patients are complex wholes in terms of mereological approach and in terms of emergent approach. Because the interaction between different dimensions of patients are not hypothesized and then be tested. Thus, patients are implicitly treated as merely the sum of constituent parts, though the emergent approach is adopted in the W&C model. This is an obstacle of the implementation of PCC since the result of clinical research is similar to the consequence of adopting reductionism. I will argue that it is a problem and the W&C model is responsible to the causal bias.

Bakas et al. (2012) reviewed fourteen articles that use the W&C model (p.8). Nine of them generated the hypotheses of bottom-up causation. None of them is about hypotheses regarding top-down causality or causation in other directions. One of them is a review article. Four of them are not relevant to hypothesis generation. I will use Frank et al. (2004) as an example. Frank et al. (2004) generated hypotheses about HRQL in patients with end-stage renal disease (ESRD) based on the W&C model. He used HRQL measurement tools such as the Short Form (36) Health Survey (SF-36) and Parfrey's health questionnaire for ESRD to measure the change in level two and level three HRQLs. The level one HRQL was measured by "patients' most recent laboratory tests" (Frank et al., 2004, p.10). The following are the generated hypotheses:

H1) Quality of life of ESRD patients will be lower than that of the general population on all dimensions.

H2) Quality of life of ESRD patients will be related to biological and physiological factors.

H3) The more numerous and severe patients' symptoms, the lower their quality of life

will be.

H4) Patients in the pre-dialytic stage will report a lower quality of life on all dimensions. Patients receiving dialysis will report higher quality during the first year, while those who have been on dialysis for more than one year will have lower quality of life on all dimensions.

H5) Patients' individual and environmental characteristics will be related to their quality of life, so that quality of life will be higher among men, younger patients, those who are better educated, are employed and have higher socio-economic status.

According to Frank et al. (2004), "[t]he dependent variable in this study was health-related quality of life, defined as patients' reports of their level of functioning and well-being during the past four weeks." And, "[i]ndependent variables included symptoms, biological and physiological measures, and patients' individual and environmental characteristics, ..." (Frank et al., 2004, pp.9-10). Independent variables involve level one and two HRQLs and individual and environmental characteristics in the W&C model. The dependent variable involves level three and four HRQLs in the W&C model. Next, I will use the term 'dependent variable' to refer to level three and level four HRQLs. 'Independent variable one' refers to level one HRQL. 'Independent variable two' refers to level two HRQL. 'Independent variable three' refers to individual and environmental characteristics.

In what follows, I transform those hypotheses in terms of dependent and independent variables:

H1') The change of independent variable three causes the change of the dependent variable. (See Figure 3.2)

H2') The change of independent variable two causes the change of the dependent variable. (See Figure 3.3)

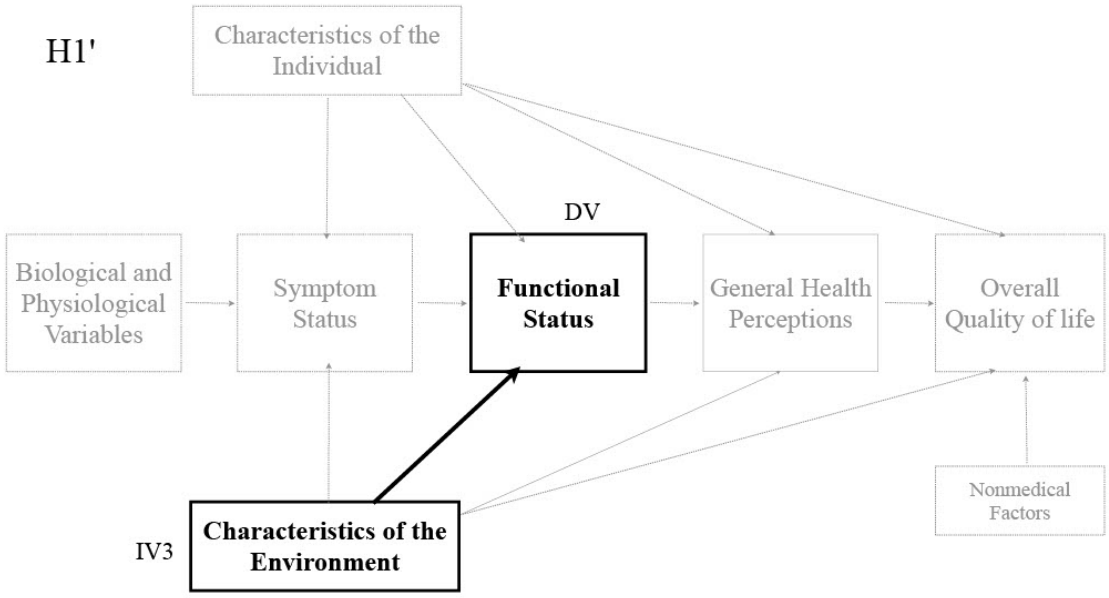

Figure 3.2: The First hypothesis in Frank et al. (2004). The description of arrows are omitted.

H3') The change of independent variable one causes the change of the dependent variable. (See Figure 3.4)

H4') The change of independent variable two causes the change of the dependent variable. (See Figure 3.5)

H5') The change of independent variable three causes the change of the dependent variable. (See Figure 3.6)

As we have seen, there are only hypotheses that involve the bottom-up causation of HRQLs if we focus on the relationship between HRQLs. Although the relationship between individual and environmental characteristics and the dependent variable could be regarded as not a bottom-up causation, it is still indicated that the changes in higher-level HRQLs are effects of the changes in lower-level HRQLs. In other words, in Frank et al. (2004), the lower-level of HRQLs are the roles that cause the change while the higher-level HRQLs are the roles that are caused to be changed. In the other eight pieces of research, a similar situation was found.

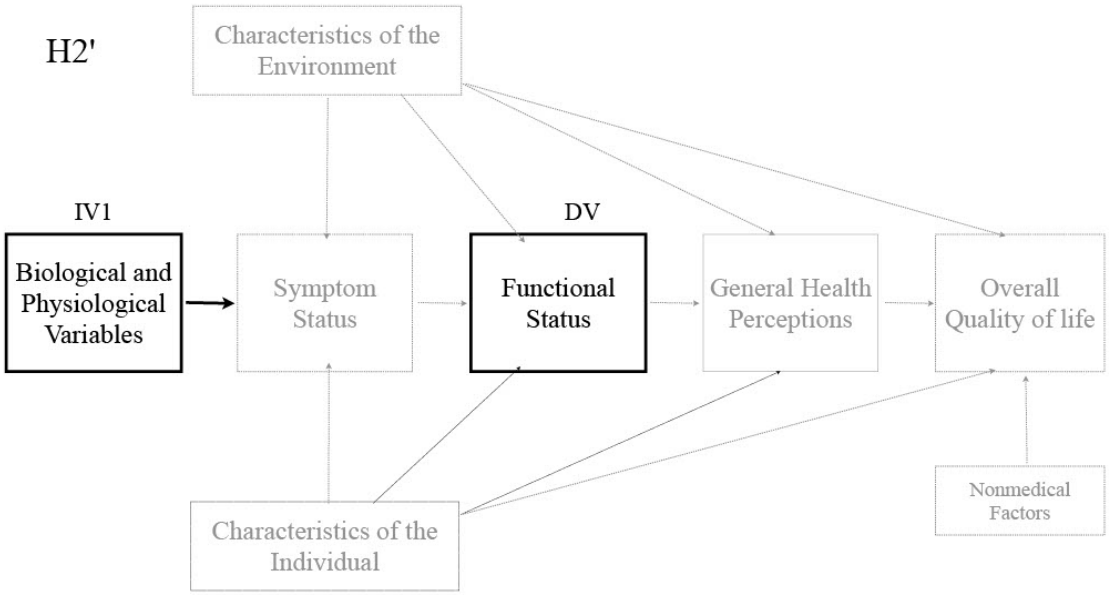

Figure 3.3: The second hypothesis in Frank et al. (2004). The description of arrows are omitted.

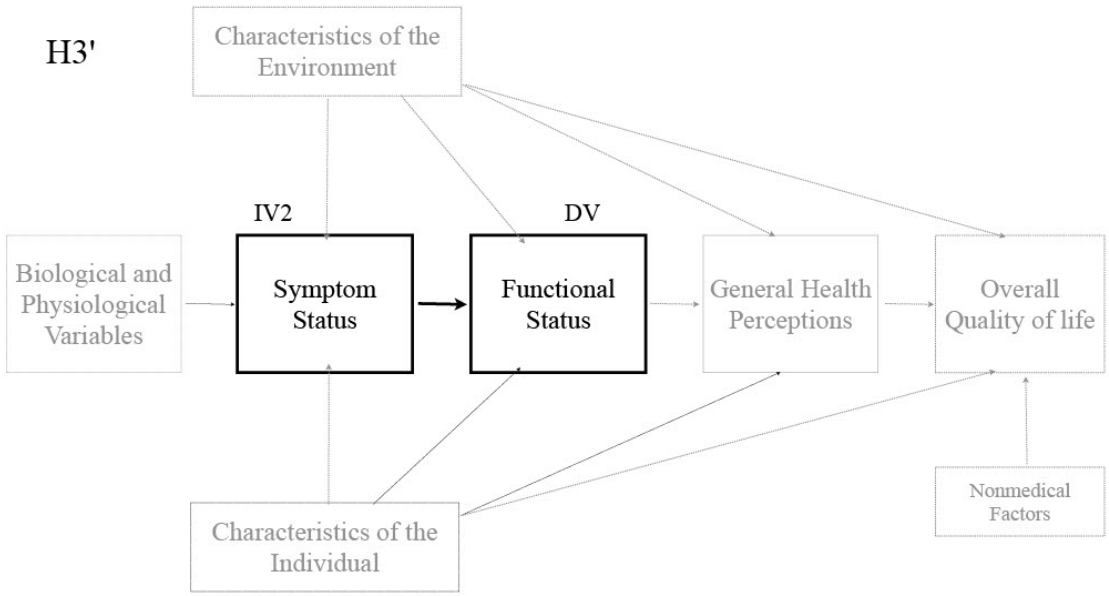

Figure 3.4: The third hypothesis in Frank et al. (2004). The description of arrows are omitted.

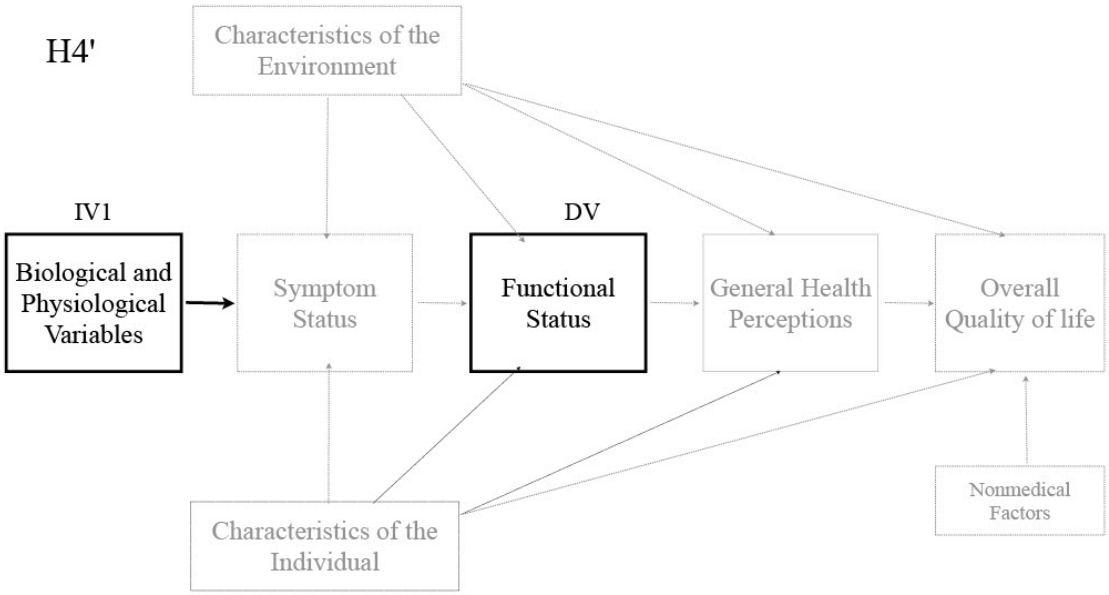

Figure 3.5: The Fourth hypothesis in Frank et al. (2004). The description of arrows are omitted.

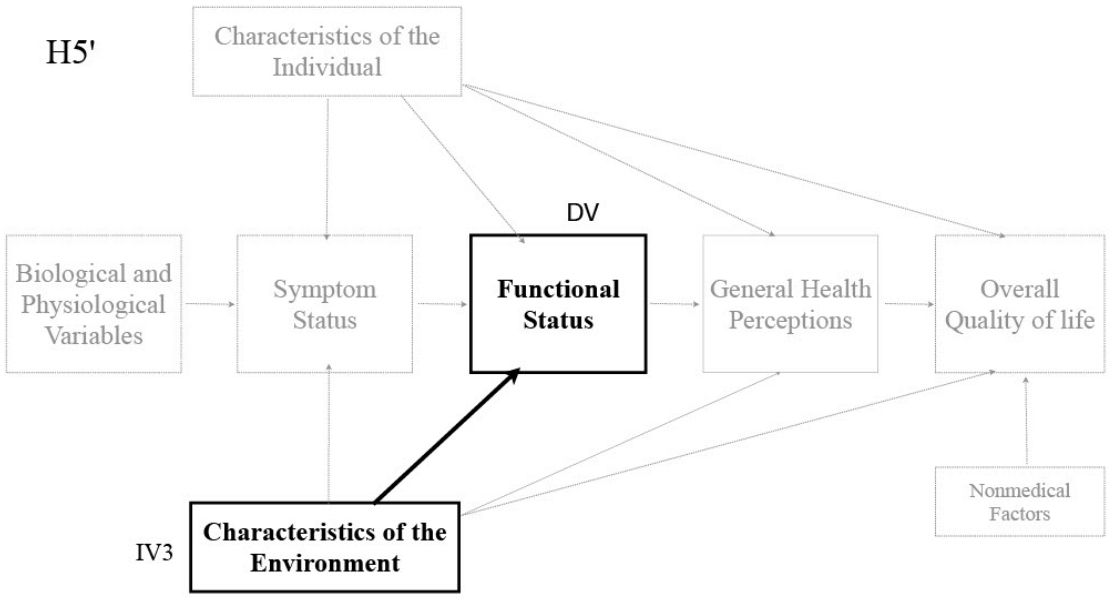

Figure 3.6: The Fifth hypothesis in Frank et al. (2004). The description of arrows are omitted.

Hypotheses generation is crucial to identify how clinical about causation in the HRQL theoretical model gives a way to think about the relationship between different levels of HRQLs. The ontological assumptions about causation in the HRQL theoretical model give a way to think about the relationship between different levels of HRQLs. And then, this would be the starting point for generating hypotheses. I have mentioned that the starting points of hypotheses generation with mereological approach are different from those with emergent approach. I have also argued that the emergent approach is adopted in the W&C model. So, it is supposed that the interaction between constituent parts of patients are considered when generating causal hypotheses about the changes in patients as complex wholes. However, the scenario we have seen in Frank et al. (2004) is not what we expected. We expected that not only bottom-up causality but also top-down causality or causation in other directions are investigated in a causal inquiry. However, the W&C model offers the starting point of causal inquiry with mereological approach. The hypothesis generation is thus misguided to ignore top-down causality or causation in other directions. I have mentioned several works that aim to contribute to the implementation of PCC (Rocca and Anjum, 2020; Anjum, 2016). Wilson and Cleary (1995) also tried to incorporate quality of life, i.e., they were not satisfied with healthcare care that ignore non-biomedical factors as causes of the change in HRQLs. (Wilson and Cleary, 1995). These works show the expectation that the proposed theoretical framework would contribute to a change in healthcare practice. In other words, the change in the practice of healthcare that promotes PCC is the ultimate goal of proposing or invoking a HRQL theoretical model in clinical research. However, causal hypotheses with the kind of causal bias have little to do with the interdependence of constituent parts of patients as complex wholes. And then a question is raised: If the W&C model does not contain the two ontological assumptions about causation, will clinical researchers generate causal hypotheses in the way like Frank et al. (2004)? Hard to say no. If this is the case, the ultimate goal is hard to be achieved since invoking the W&C model does not guide clinical researchers generate causal hypotheses that consider the interaction and interdependence between parts of patients.

One way to block this worry is to require centering the emergent approach in the HRQL theoretical model. By doing so, top-down or the other directions of causation are also expected to be investigated in clinical practice as much as bottom-up causation. There will be a clear starting point, rather than an implicit endorsement that is easy to ignore, like which we have seen in the W&C model.

But one might still ask, is the W&C model responsible for the bias in hypothesis generation? I will give several reasons to answer yes. The first reason is that the W&C model gave the start point for clinical research. As a tool, the W&C model provides a framework for generating hypotheses in Frank et al. (2004), for instance. Frank et al. (2004) used the level one and two HRQLs as "predictors of quality of life of ESRD patients" (p.4). Without the conceptual framework, we can hardly say that Frank et al. (2004) would generate the same hypotheses. The other reason is that the relationships between HRQLs are not clear in the W&C model. The function of the description of arrows in the W&C model is merely to refuse two thoughts: 1) it is impossible that there is a reciprocal relationship between the HRQLs and 2) it is impossible that there is a relationship between HRQLs if there is no arrow between them. However, how should we address the reciprocal relationship between the HRQLs and relationships between HRQLs if there is no arrow between them? The description of arrows is unclear, and it may not be able to provide practitioners with a way to assume causal relationships between HRQLs, except for bottom-up causations. In sum, the W&C model is not capable to provide clinical research with a starting point to investigate bottom-up causality or the other directions of causation. Yet it was supposed to do so and it was invoked. Hence I conclude that the W&C model is responsible for the causal bias.

I have shown why and how the W&C model instills the causal bias into clinical research. I provided the case of Frank et al. (2004) as a concrete example. If emergent

approach is centered in the HRQLs theoretical models, then at least practitioners would get a starting point to generate hypotheses of the other directions of causation. Thus, investigating interdependence and interaction is enabled. In the next section, I will consider some proposals that avoid the disadvantages of the W&C model, while the two ontological assumptions about causation play significant roles.

4. Revising the W&C model

I have argued the W&C model instills the causal bias, and it is responsible to the causal bias. Yet only to point out the problem is not enough, I will also offer a solution. In this section, I will utilize a existing revision of the W&C model and some philosophers' insights to propose a revised version of the W&C model.

4.1 The Existing Revision of the W&C Model

Bakas et al. (2012) recommended the revised version of the W&C model, which was proposed by Ferrans et al. (2005) because it "provides clear conceptual and operational definitions, and it also clarifies relationships among concepts to guide research and practice" (p.10). Ferrans et al. (2005) deleted the characterization of the arrows, and the non-medical factors as one factor that influences HRQLs. They added the arrows from individual and environmental characteristics to level one HRQL (See Figure 4.1). The revised version makes level one HRQL could also be the effects in causation. Yet, it still retains that arrows represent dominant causal associations, and my worry remains.

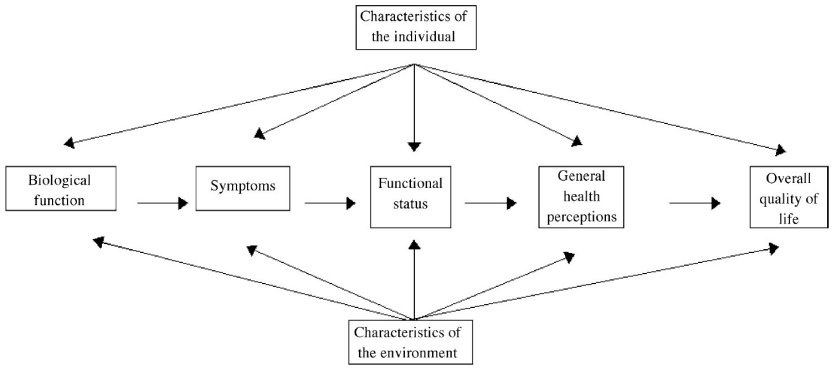

Figure 4.1: The HRQL theoretical model proposed by Ferrans et al. (2005). Reproduced from Ferrans et al. (2005).

4.2 Revised W&C Model

In order to remove the identified causal bias that implicitly in the W&C model, I will revise the model in the following two ways: 1) Center emergent approach and 2) the other directions of causation should weigh as much as bottom-up causation in the first place.

The Figure 4.2 is the revised model. Every variables in the W&C model is retained except non-medical factors. The original descriptions are replaced by descriptions in Ferrans et al. (2005). The arrows are replaced with dotted lines. I plot HRQLs, individual characteristics, and environmental characteristics into a topological space. All of them connect with each other with dotted lines. By doing so, no causal association is dominant in advance nor any relationship between all variables is established in advance. The dotted lines represent a place where the relationships between variables could be hypothsized. Relationships such as correlation or causation can be assigned according to the needs of practitioners, or one day when the results of clinical research of a given group become a consensus, we can assign a relationship to these dotted lines to generate hypotheses. So this model could be plural and variant in different issues or groups.[1] For example, my proposal can be used in research on ESRD patients and in palliative care research. It is possible and natural that the relationships assigned to the former and to the latter are different, since the situation of ESRD patients and palliative care patients is probably not the same.

What is it like to use the revised HRQL theoretical model to generate causal hypotheses? Imagine that we are the clinical researchers that investigating HRQL of patients with ESRD. When we use the revised model, the starting point of the causal inquiry is that there

[1] Alexandrova (2017) discussed well-being in detail. She thought HRQL is a concept of well-being while it should be distinguished from subjective well-being, for instance. She showed how to develop a mid-level theory of well-being to provide practitioners with a useful toolbox (Alexandrova, 2017). She did concern about how philosophical theories can be good tools for practice, which is a valuable insight to revising the W&C model.

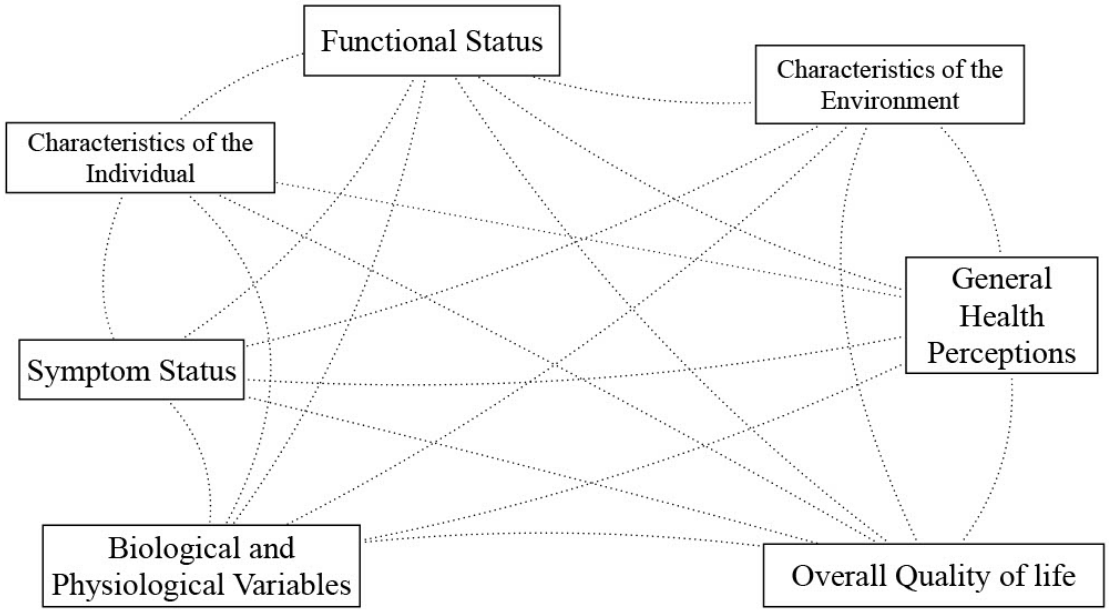

Figure 4.2: The revised HRQL theoretical model

are complex relationships between different HRQLs. So there are many potentials to be generated rather than intuitively generating bottom-up causal hypotheses. The existing results of clinical research, the concrete situation about the available tools or subjects in our project, etc., can be the materials to customize the causal hypothesis generation. The consequence can be shown by plotting indicators of relationships. For example, if we found the change in general health perceptions of patients with ESRD has causal power to the change in symptom status, then we plot an arrow to replace the dotted line which links these two. The revised model avoids the causal bias because there are no 'dominant causal association' posited in the model. Instead, clinical researchers will admit there can be causation in different directions in the first place when they use the revised model.

5. Conclusion

I focus on the ontological assumptions about causation in PCC and the HRQL theoretical model. It is indicated that debating the causal ontology can be a procedure that positively influences the healthcare that promotes PCC. I argued that the ontological assumptions about causation in the W&C model and PCC were consistent. Yet, there is a causal bias of the W&C model. Clinical researchers who use the W&C model generate bottom-up causal hypotheses far more frequently than the other kinds of causal hypotheses. The consequence can be seen if the clinical researchers use reductionistic HRQL theoretical model, although the emergent approach is adopted in the W&C model. Hence, patients are implicitly treated as merely the sum of their parts. However, the interaction between constituent parts of patients is considered in the healthcare that promotes PCC. I argue the W&C model is responsible for the causal bias since it does not provide clinical researchers with a starting point for hypothesis generation that considers the interaction between parts of patients. To avoid the causal bias, I propose a way to revise the W&C model by 1) centering the emergent approach, and 2) making the other directions of causation weigh as much as bottom-up causation in the first place. The revised model retains the basic structure of the W&C model and respects the needs of clinical research. By doing so, I have made the first step that influences the practice of healthcare that promotes PCC through analyzing the ontological assumptions about causation.

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