國立陽明交通大學
心智哲學研究所
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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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中華民國 一一一年七月
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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

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摘　　要

以病患為中心的照護 (patient-centered care,PCC) 是一個重視病人喜好、需要與自主的照護取向。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為評估 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) 的理論模型為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) 發展了最為廣泛使用的 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身體功能、生理條件與經濟因素等等條件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，我為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，以病人為中心的照護，因果本體論，因果複雜性。

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中文論文綜論

以病患為中心的照護 (Patient-Centered Care, PCC) 是一種備受重視醫療照護的取向。秉持 PCC 的照護者們重視病人的喜好、需要與自主。要如何評估照護的品質? 或者要如何評估提供給病患的醫療照護確實有落實 PCC 的精神? 測量病患的與健康有關的生活品質 (Health-Related Quality of Life, HRQL) 是評估的重要指標之一。除了描繪 PCC這個備受重視的照護取向，在第一章中我刻劃了 HRQL 的概念生活品質被認為是與幸福 (Happiness) 或者生活滿足感 (Life Satisfaction) 相關聯的概念 (Fayers and Machin, 2002; Alexandrova 2017)，但生活品質可能會受到非常多因素的影響，如政治、社會、心理、經濟等等條件。本文僅考慮在醫療照護的脈絡之下，與健康有相關的生活品質的層面，換言之，HRQL 就是與健康狀況有關聯的生活品質。為了更清楚的說明 HRQL 的概念，將卡諾夫斯基量表 (Karnofsky Performance Scale, KPS) 是有幫助的。KPS 是一個由醫生報告的量表，這個量表評估了病患的健康功能，包含生活自理、駕駛、可負荷的工時等等；總分為 100 分，最低為零分，分數越高代表受測者的健康狀況越好。KPS 被認為是第一個將非生理學因素納入健康狀況評估的工具 (Karnofsky and Burchenal, 1952; Fayers and Machin, 2002, p.7)。在二十世紀下半葉，KPS 被拿來測量接受化學治療或者苦於慢性病的病患的健康狀況。Timmerman (2012) 也討論了 KPS 的歷史，用以作為探究為何測量生活品質會逐漸被重視的案例研究。KPS 做為一個 HRQL 測量工具，展示了在 HRQL 的測量實作中，並不僅有生物醫學因素如血壓、心跳速率等等被納入考慮，非生物因素如病患是否能正常進行日常活動也同樣被當作病患的生活品質是否好或壞的依據。簡言之，HRQL 就是被 KPS 這類同時測量了生物醫學因素與非生物醫學因素的工具所測量的構念。

McClimans (2021) 試圖建立 HRQL 與 PCC 之間的緊密關聯，她梳理了 HRQL 和 PCC 的歷史，並主張 HRQL 測量工具是落實 PCC 的載體。他認為 HRQL 應該與其他安適

(well-being) 的概念有所區分，因為 HRQL 測量工具逐漸受到歡迎是因為其在捕捉病患的主觀經驗的突出表現，並且，病患的主觀經驗在 PCC 這個取向中尤其重要。同時，其他安適的測量工具並沒有如 HRQL 測量工具一樣與 PCC 共享相同的脈絡，他們要不是群眾等級的測量，要不僅關注病患的能力 (capability) (McClimans, 2021)。可以這麼詮釋 McClimans (2021) 的想法作為 PCC 的載體，探究 HRQL 的臨床研究的目標是使 PCC 能更落實。我將這個想法拓展，並概念化了 HRQL 的測量實作。首先，臨床研究者們調用關於 HRQL 的概念模型以生成與 HRQL 相關的假設。例如 Frank et al. (2004) 調用了 Wilson & Cleary (1995) 提出的 HRQL 概念模型，生成了幾個關於末期腎臟病患者不同的健康條件之間關係的假設，意圖透過檢驗假設來探究這些不同的健康條件如何互相影響。接著，臨床研究者會依據需要來選擇合適的 HRQL 測量工具來測量 HRQL，以便檢驗假設。Frank et al. (2004) 便使用了包括健康調查量表 (The Short Form (36) Health Survey, SF-36)、使用儀器測量得出的血紅蛋白含量等測量工具來測量病患的生物醫學指數、症狀的狀態及功能狀態等。這些臨床研究的結果能幫助照護的實作者們評估 PCC 這種照護取向在某一個接受醫療照護的群體中被落實的如何。簡言之，HRQL 的概念模型提供生成假設或挑選 HRQL 測量工具的資訊與指示給臨床研究者，接著臨床研究者依據需要來使用 HRQL 測量工具，最後研究的成果回過頭來幫助評估醫療照顧的品質。在我概念化的 HRQL 測量實作裡，HRQL 有三種意涵被測量的構念，概念模型，以及測量工具。McClimans (2021) 所討論的幾乎專注於 HRQL 測量工具，對 HRQL 概念模型的討論甚少，但是我在本文中主張，McClimans (2021) 所建立起的 HRQL 測量工具與 PCC 之間的緊密關聯，同樣也出現在 HRQL 概念模型與 PCC之間，從我的概念化中，可以看到關於 HRQL 的臨床研究頻繁使用 HRQL 概念模型，若要說 HRQL 概念模型在評估 PCC 裡並未扮演重要角色，那麼 HRQL 的測量實作就無法被給出合理的說明。那麼，如此一來，分析 HRQL 概念模型的預設對於 HRQL 的測量實作就是有意義的工作。本文將以Wilson & Cleary (1995) 提出的 HRQL 概念模型 (W & C 模型) 當作探究的對象之一，分析這個概念模型的以及 PCC 的關於因果的本體論預設。W&C 模型是目前最被廣泛使用在 HRQL 臨床研究的概念模型 (Bakas et al., 2012)。經過我的分析之後，我主張 W&C 模型與 PCC 兩者的關於因果的本體論預設是相容的，但是在臨床研究的案例中，我發現了一種因果偏誤，即調用 W&C 模型的臨床研究大量

生成生物醫學因素會影響非生物醫學因素的假設，而非生物醫學因素會影響生物醫學因素的假設則很少會生成。若是如此，那麼 W&C 模型就隱晦地引導醫療照護實作忽略病患的非生物醫學因素如何影響生物醫學因素，進而負面的影響秉持 PCC 取向的醫療照護。因為僅有關於生物醫學因素如何影響病患的 HRQL 的資訊的話，病患的喜好、需要與自主或其他非生物醫學因素會如何影響病患的 HRQL 便是不確定的 (uncertain)，進而 PCC 也難以被落實。

那麼，PCC 關於病患的本體論預設是甚麼呢? 首先，病患是一個複雜的整體 (a complex whole)，許多與健康有關聯的因素還有他們的互動都會影響病患的 HRQL。在科學哲學的脈絡中，至少有兩種方式來理解複雜的整體的意義，第一是分體論的取向 (mereological approach)，另一個是突現的取向 (emergnet approach) (Rocca and Anjum, 2020)。一部汽車可作為分體論取向的例子：我們想像一部由引擎、輪胎、車窗等等部分組成的車子，車子是複雜整體，因為有許多不同的零件組成了這台車子。不過，這些零件相加等於這台車子，並沒有甚麼現象能說明這台車子比其零件的組合還要多出甚麼。接著，這台車子的出了一些問題，所以我們送它進廠維修。經過一番檢查，發現是引擎出了問題。在進廠維修的過程中，我們去探究一個問題是甚麼造成了這個不幸的事件? 既然我們接受這台車子僅僅是零件的總和，那麼我們探究這個問題的方式就是一個一個零件去檢查，當我們發現確實有某些零件出問題的時候，就可以說這些零件是車子故障的原因。另一方面，突現取向的例子可以是河狸所居住的環境。這個環境包含了河狸群體以及周遭的河水、河岸或岸上的植被樹木等等，光是河狸以及植被或河水，並不足以構成這個環境，因為河狸與這些周遭不斷的互動，如他們建造水壩，周遭的各種條件對河狸進行天擇等等。確實這個環境的組成部件有植被、河水、河狸等等，但當他們一起構成了這個環境，這些組成部件之間的互動使得這個環境大於植被、河水、河狸等等的總和。若我們要探究為甚麼河狸的數量減少時，我們很難僅考慮是否是河水水位下降，還是河狸的傳染病等等因素，而必須將整個環境中組成部件的互動一起考慮進去。換言之，突現取向的複雜整體的改變，需要考慮所有部件的互動，而非將組成部件一個一個分開檢驗。現在我們可以問 PCC 將病患是為哪一種取向的複雜整體? 我主張是突現的取向。理由可以從一個問題去分析提供 PCC 取向的醫療照護時，病患的組成部件是否被一個一個分開來檢驗? 從 PCC 的內容和 Lusk (2013) 的分析來看，提供醫療照護時，每一個病患的背景與脈絡都被加以考慮。既然是從脈絡和背景出發，那麼不僅僅是分別考慮病人的經濟條件、心理條件就提供照護，而是要將這些條件之間的互動一起考慮，才能決定對病患來說最好的照護決定是甚麼。由此我結論 PCC 將病患是為突現取向的複雜

整體。

接著，有兩個核心的因果本體論預設可以被推導出來 1) 病患的組成部件是互相依賴的，和 2) 不同維度的改變可以共同造成病患的改變。互相依賴的意思是，我們必須同時考慮從生物醫學因素到非生物醫學因素的因果假設，以及從非生物醫學因素到生物醫學因素的因果假設。Rocca and Anjum (2020) 區分了兩種因果自下而上的和自上而下的。「自下而上的」因果即生物醫學因素的改變會導致非生物醫學因素的改變，「自上而下的」則是相反的方向。接下來我會用這組詞彙來對因果方向進行分析。現在我要考慮 W&C 概念模型包含的因果本體論預設，檢驗是否與 PCC 的因果本體論預設相容，來考慮我們在這個被廣泛使用的模型是否確實的幫助了落實 PCC。

W&C 概念模型包含了五個等級的 HRQL，環境及個人因素，非醫學因素，還有這些構件之間的關係所組成 (參考 Figure 3.1)。等級一的 HRQL 為病患的生理學及生物學變項，依序到等級五為症狀狀態，功能狀態，整體的健康感受，整體的生活品質。等級越低則越容易定義與測量，等級越高則越複雜，越不容易定義。環境因素跟個人因素並非 HRQL，但是會影響 HRQL。概念模型中的箭頭代表主要的因果關係 (dominant causal association)。從症狀狀態指向功能狀態的箭頭就代表前者的改變會造成後者的改變。在給出 W&C 概念模型的基本描繪後，我將問三個問題來檢驗 W&C 概念模型和 PCC 的因果本體論預設是否相容 1)W&C 概念模型是化約論的模型嗎? 2)W&C 概念模型只包含了自下而上的因果關係嗎? 3)W&C 概念模型是否包含了 PCC 的兩個核心的因果本體論預設? 只要前兩個問題的答案是肯定，那麼W&C 概念模型與 PCC 的因果本體論預設就是不相容的。另外，若第三個問題的答案是肯定的，那麼我們可以確定 W&C 概念模型的因果本體論預設有包含 PCC 的因果本體論預設，我們也就不必擔心 W&C 概念模型包含不合適的因果本體論預設。

我從第一個問題開始。化約論認為所有事件和過程都必須是物理原因所造成的結果(Rocca and Anjum, 2020)。但是在 W&C 概念模型的描述裡面，我們可以看到在整體健康感受和總體生活品質之間有一個箭頭，而兩者都包含了非物理的因素，也就是說，我們可以確定在 W&C 概念模型中包含了非物理的因素作為因果關係的原因，所以，W&C 概念模型並不是一個化約論的模型。接著我們追問，那 W&C 概念模型中的因果關係是否都是自下而上的? 我們可以從 Wilson and Cleary (1995) 中對箭頭的描述找到答案根據他們的描述，箭頭所表達的是「主要的」因果關係，箭頭不表示沒有雙向的關係，沒有箭頭的兩個因素之間也未必沒有任何相關。還有另一個方式能給第二個問題否定的答案 Wilson and Cleary (1995) 提供了關於攝護腺疾病的臨床研究案例，一些病人報告說他們並未因症狀變得嚴重而使得整體健康感受有所改變，也沒有因為症狀更嚴重而使得生活的自理功能受到影響。如果 W&C 概念模型只有自下而上的因果關係，那麼我們不可能遇到等級較低的 HRQL 改變，但是等級較高的 HRQL 卻未改變的情況。第二個問題的答案也是否定的。最後我主張 W&C 概念模型確實有包含 PCC 所包含的兩個核心因果本體論預設。首先，根據對箭頭的描述，不同 HRQL 之間的雙向關係的可能性並未被排除，Wilson and Cleary(1995) 也確實考慮了不同 HRQL 之間的互動，而並非主張箭頭所指的方向就是唯一的因果方向；另外，我們可以看到同時有多個不同的因素會影響病患的功能狀態。兩個核心的因果本體論預設在 W&C 概念模型裡面都可以被推導出來。

不過，我們分析 HRQL 概念模型的目的是為了影響實作，也就是說，如果在臨床研究的實作中我們沒有看到預期的結果時，那麼 HRQL 概念模型就可能有其問題。我想指出 W&C 概念模型向臨床研究者隱晦得灌輸了某種因果偏誤。對臨床研究者而言，生成自下而上的因果關係的假設是非常自然的，但是我們仔細看臨床研究中的假設就會發現，自上而下的因果關係的假設卻非常少被生成甚至檢驗。若是如此，臨床研究的結果就沒辦法為醫療照護實作提供關於自上而下的因果關係的指示或資訊，那麼病患被視為分體論取向的還是突現取向的複雜整體就事實上沒有區別，同時臨床研究只提供了與自下而上的因果關係有關的指示與資訊，病患也就隱晦的被視為分體論取向的

複雜整體，進而 PCC 無法被落實。Bakas et al.(2012) 回顧了 14 篇臨床研究，我逐個檢視這 14 篇臨床研究所生成的假設。其中有 9 篇臨床研究生成了自下而上的因果關係的架設，4 篇與生成假設無關，1 篇為回顧性的文獻。我將提供其中一篇，即 Frank et al., (2004) 所做的臨床研究做為案例，並論證 W&C 概念模型是這種因果偏誤的主因。

Frank et al.,(2004) 生成了末期腎臟病患的 HRQL 有關的假設，並使用 SF-36 及帕佛瑞健康調查 (Parfrey's health questionnaire) 等 HRQL 測量工具對 W&C 概念模型中的等級二、等級三的 HRQL 進行測量。病患的等級一的 HRQL 則是病患的實驗室檢驗數據 (Frank et al., 2004, p.10)。這個臨床研究的依變項是病患的功能狀 (等級三的 HRQL)，有三個自變項：生理與生物學變項、症狀的狀態、環境或個人因素，我分別用自變項一、自變項二、自變項三來稱呼這三個變項。他們生成了五個假設，為了方便，我直接轉化為以變項來表達的形式：

H1') 自變項一和自變項二的改變造成依變項的改變。(參閱 Figure 1)

H2') 自變項一的改變造成依變項的改變。(參閱 Figure 2)

H3') 自變項二的改變造成依變項的改變。(參閱 Figure 3)

H4') 自變項一的改變造成依變項的改變。(參閱 Figure 4)

H5') 自變項三的改變造成依變項的改變。(參閱 Figure 5)

我們可以看到，在這個臨床研究中，在 HRQL 的互動之間幾乎只有自下而上的因果關係假設被生成，很少自上而下的因果關係假設被生成。在 Bakas et al.(2012) 所提及的其他 8 篇頻床文獻中，也都能找到類似的情況。

調用 HRQL 概念模型是生成假設的起點，也就是說，HRQL 概念模型提供了臨床研究者一個對 HRQL 構念的基本架構，這個 HRQL 概念模型所包含的因果本體論預設也是到底什麼樣的因果關係假設會被生成的出發點。已有一些文獻並不滿意忽略了自上而下的因果關係的醫療照護而做出努力，這些文獻展示了概念模型能夠為改變實作做出貢獻的期待 (Rocca and Anjum, 2020; Anjum, 2016; Wilson and Cleary, 1995)。但是當我

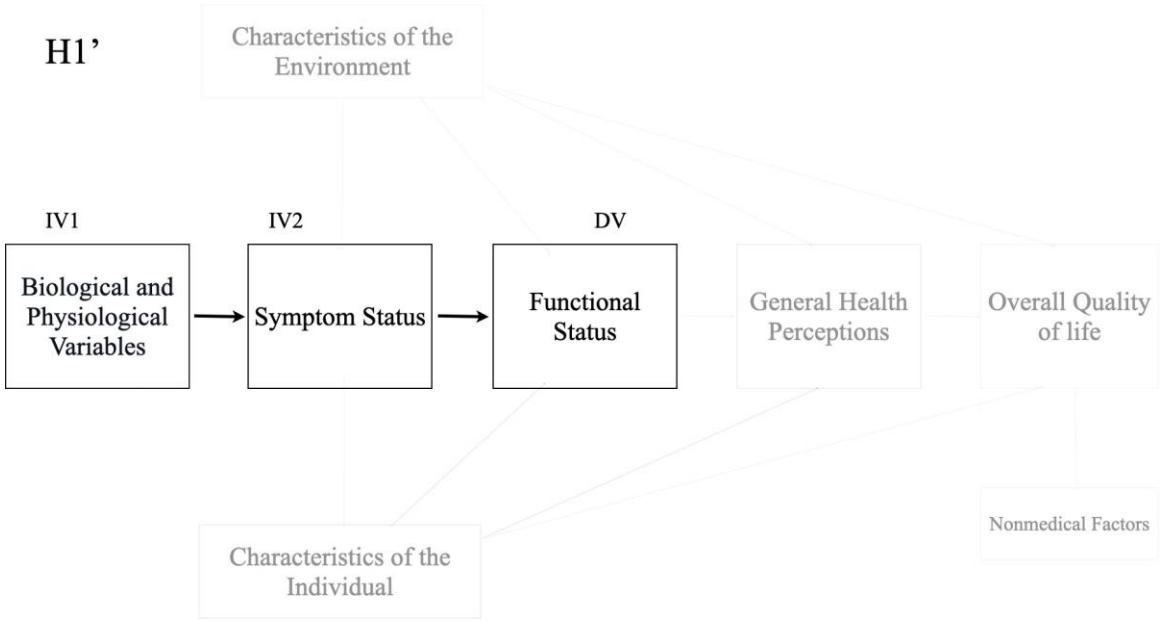

Figure 1: The First hypothesis in Frank et al. (2004). The description of arrows are omitted.

們把鏡頭轉移到臨床研究的時候，卻發現自上而下的因果關係假設很少被探究，進而病人不同條件與組成部件之間的互動並沒有在臨床研究中被考慮。一個問題就出信了：如果今天 W&C 概念模型並沒有包含 PCC 的兩個核心因果本體論假設，那麼調用 W&C概念模型的臨床研究是否仍僅僅關注自下而上的因果關係架設? 我認為很難說不。也就是對於概念模型所做出的進步與改變並未成功的去影響實作。如果影響實作是對概念模型進行分析的最終目標，那麼現在我們必須對 W&C 概念模型在進行一些修改。一個避免這個因果偏誤的方式是將突現取向的因果本體論預設在概念模型中更被強調，這樣的話，在生成假設的起步就有更清晰的動機或起點能夠生成自上而下或其他方向的因果假設，而不只有自下而上的因果關係假設。

仍然有人會問，W&C 概念模型就是造成這種因果偏誤的主因嗎? 我會提供幾個理由說答案是肯定的。第一，我們知道 W&C 概念模型是生成假設的起點，這點在我概念化的測量實作中已有說明。Frank et al.(2012) 也說明等級一與等級二的 HRQL 在他們的研究中被當作病患 HRQL 的預測因子 (p.4)。若是沒有調用 W&C 概念模型，連要如何生成假設都將模糊不清。正是 W&C 概念模型提供了一個 HRQL 的概念框架，臨床研究者們才能使用「生理與生物學變項」、「症狀狀態」等概念作為假設當中的變項。另一

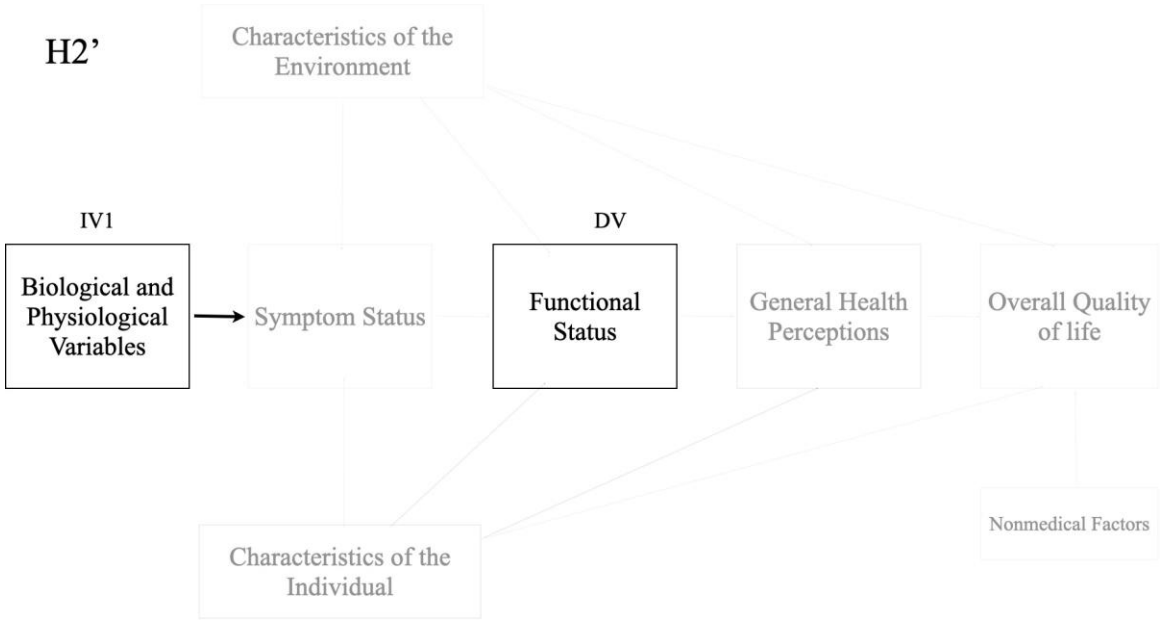

Figure 2: The second hypothesis in Frank et al. (2004). The description of arrows are omitted.

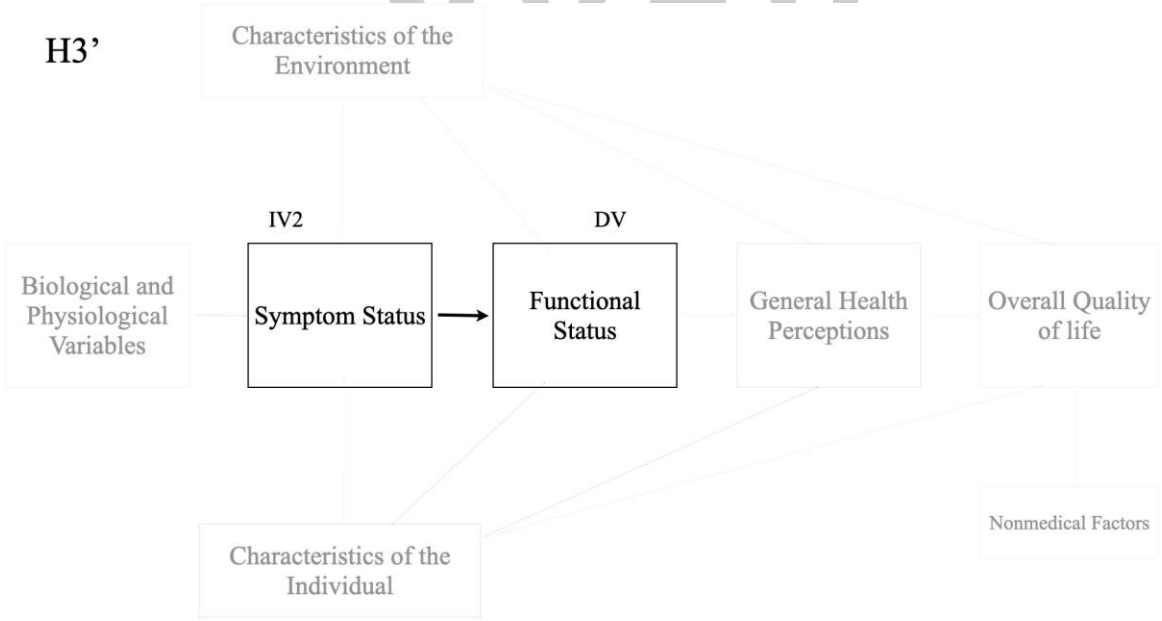

Figure 3: The third hypothesis in Frank et al. (2004). The description of arrows are omitted.

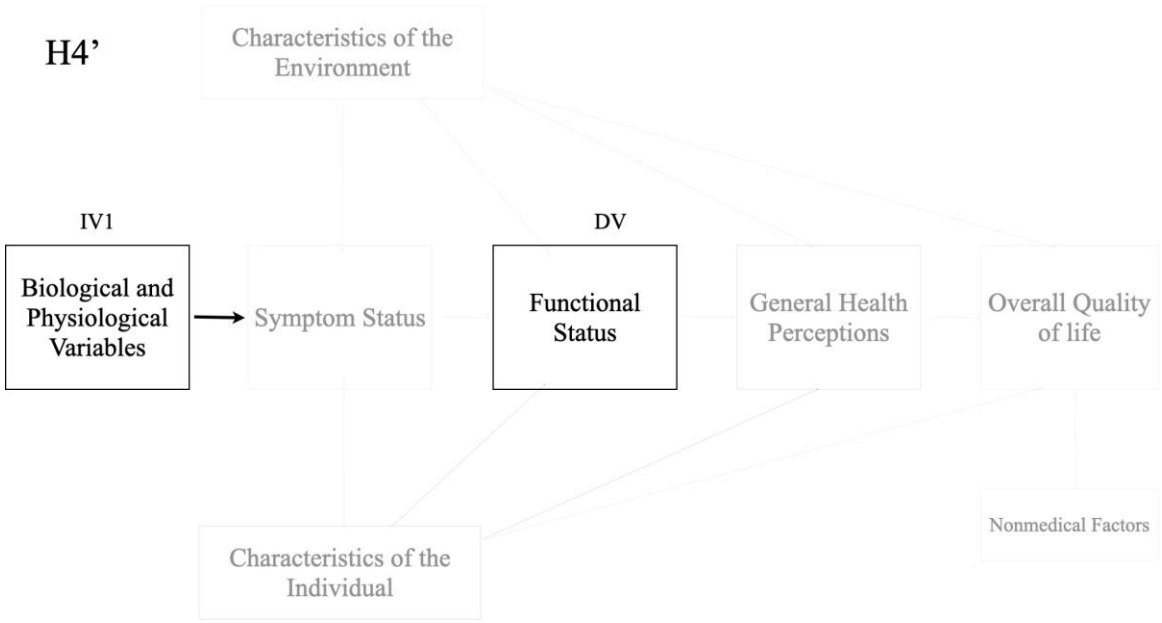

Figure 4: The Fourth hypothesis in Frank et al. (2004). The description of arrows are omitted.

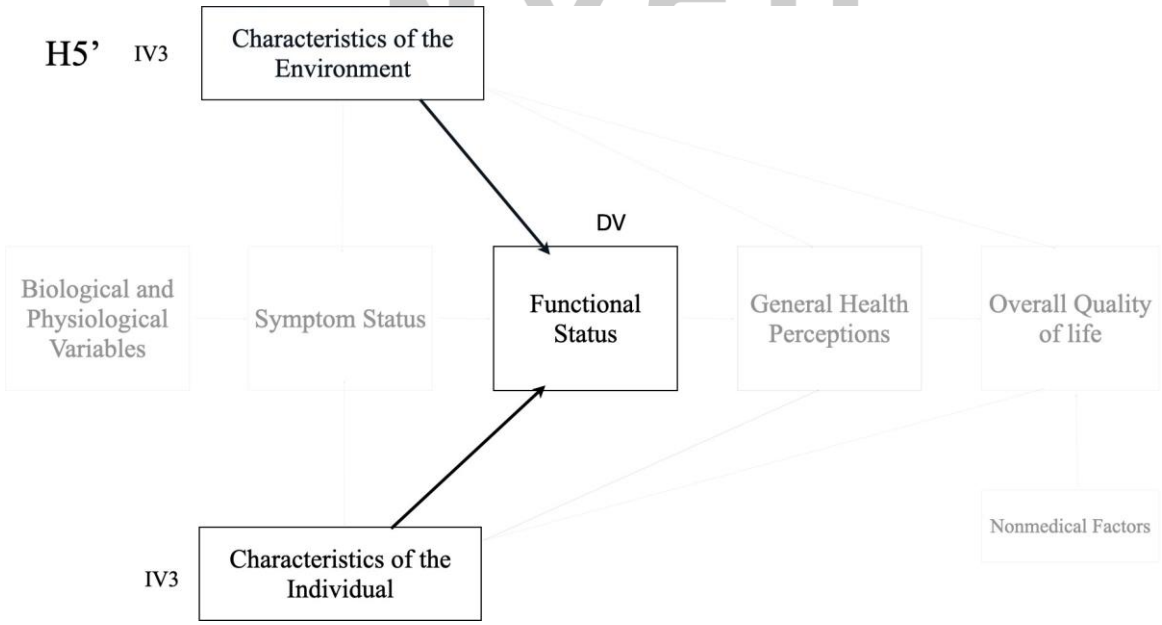

Figure 5: The Fifth hypothesis in Frank et al. (2004). The description of arrows are omitted.

個理由是，W&C 概念模型裡面不同 HRQL 之間的互動是非常模糊的。W&C 概念模型裡面關於箭頭的描述只拒絕了兩件事，第一是不可能 HRQL 之間的雙向關係，第二是沒有箭頭連接的 HRQL 之間就沒有任何關聯。這個描述雖然給 HRQL 之間的互動打開了可能性，但到底 HRQL 之間的互動是什麼樣子，可以做出甚麼樣的假設，W&C 概念模型並沒有提供足夠清楚的說明。雖然沒有箭頭連接的 HRQL 之間是有可能有相關的，但臨床研究者們到底該如何放置或猜想這樣的關係? 總之，一方面 W&C 概念模型是生成假設的出發點，同時 W&C 概念模型又在 HRQL 互動的論題上含混不清，僅在自下而上的因果關係有提供清晰的方向，我主張 W&C 概念模型因此而隱晦地向臨床研究者灌輸我所提到的因果偏誤。

不過，單就指出 W&C 概念模型的問題和因果偏誤是不足的，所以我將在本文提供一個初步的解決方案。我站在 W&C 概念模型的基礎上對其進行修改，並引入一些哲學的洞見，最後提供一個修改後版本的 HRQL 概念模型。

Bakas et al.(2012) 介紹了其中一個 W&C 概念模型的修改版本，是由 Ferrans et al. (2005) 所提出的 HRQL 概念模型。他們保留了 W&C 概念模型中的五個等級的 HRQL，環境因素與個人因素，並刪除了非醫學因素；同時，他們刪除了原有模型中對箭頭地一些描述；最後他們增加了從環境因素和個人因素指向生物學因素的箭頭。Bakas et al. (2012) 確實列出了 Ferrans 版本的 HRQL 概念模型優於 W&C 概念模型的幾個理由，但是我的擔憂在這個修改版本的 HRQL 概念模型並沒有被避免，所以我不打算考慮這個版本作為解決方案。

為了要避免我所提到的因果偏誤，我將用兩個方式來修改 W&C 概念模型：1) 將突現取向凸顯出來，和 2) 不同方向的因果關係在一開始的權重必須一樣。Figure 6 是修改後的模型。

W&C 概念模型中所有的變項除非醫學因素之外皆被保留，但是針對這些變項的說明都被取代為 Ferrans et al.(2005) 中的說明。所有的箭頭都被刪除，並且所有的變項都以虛線來連接彼此。在我的模型裡面，沒有任何一個 HRQL 是更加基礎 (fundamental)

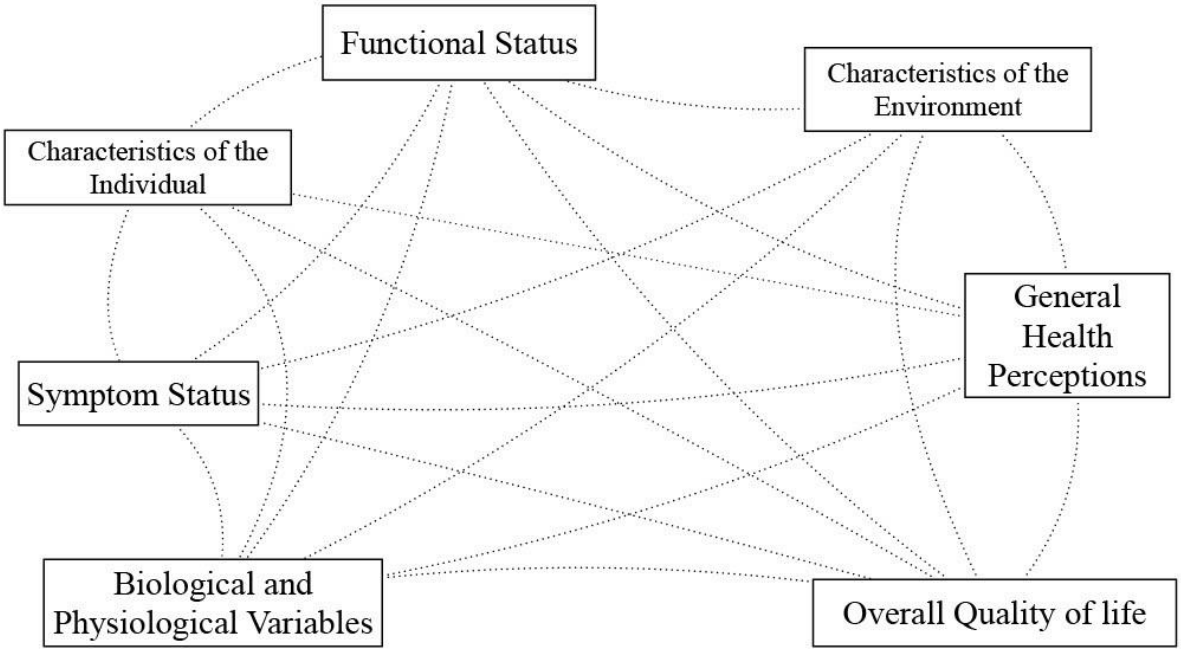

Figure 6: The revised HRQL theoretical model

的。在這個概念模型中，虛線代表的是 HRQL 之間可能的潛在相關性，相關關係或是因果關係都可以根據臨床研究的需要或實驗結果被置入某一條虛線中。所以，修改後的概念模型是多元以及變異的，在不同的臨床研究脈絡，如腎臟病末期的研究或者是臨終病患的研究，這個模型可能會提供不同的資訊給相應的臨床研究。接著我們設想，若是研究腎臟病末期的病患的臨床研究者要調用這個修改後的模型，甚麼是生成假設的起點？首先，每一個等級的 HRQL 之間都有互動的潛在可能性，並且不同 HRQL 的改變都可能會共同造成某一個 HRQL 的改變。所以，這些可能性就帶來了生成假設的彈性，反而生成自下而上的因果關係假設就不是一個直覺且自然的選擇。

在這篇文章中我聚焦於 PCC 和 HRQL 概念模型中的因果本體論假設，對因果本體論進行分析是可以對 PCC 取向的醫療照護產生正面影響的。我論證了 PCC 和 HRQL 概念模型中的因果本體論假設是相容的。但是，W&C 概念模型給臨床研究帶來了因果偏誤，調用 W&C 概念模型的臨床研究產生了大量自下而上的因果關係假設，而幾乎沒有生成自上而下的因果關係假設，這樣的結果是病患被視為分體論意義的複雜整體，而非突現意義的複雜整體，因而化約論的影子還是普遍存在於臨床照護中，但化約論與 PCC 是互相衝突的。也就是說，這樣的因果偏誤是落實 PCC 的阻礙。我論證了 W&C

概念模型是造成這種因果偏誤的主因，因為這個模型對 HRQL 之間的互動的說明非常含混，但同時此模型又是臨床研究生成假設的起點。為了要避免這種因果偏誤，我提出了一個修改方案。我的修改方案保留了 W&C 概念模型的基本架構，且尊重了臨床研究者的需要與不同的研究脈絡。透過這些分析，我做出了透過分析因果本體論的哲學工作來為醫療照護實作帶來正面影響的第一步。

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

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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

Table of Contents

List of Figures

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 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 healthcare, 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, an 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). KPS is a physician-reported outcome. It measures the functional abilities of patients that receiving the chemotherapy or suffer from chronic illness. The score would be 0-100, where 100 indicates that patients are functionally normal and 0 indicates that patients are deceased. 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-patient 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. 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 promot es 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 interaction 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).

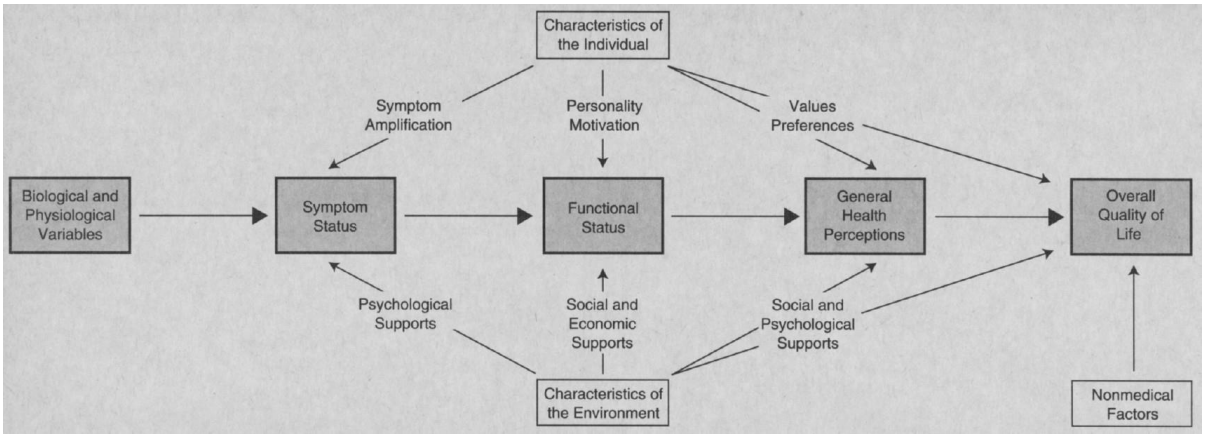

Figure 3.1: The W&C model. Reproduced from Wilson and Cleary (1995).

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 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 'DV' to refer to dependent variable, which is the functional status of patients. 'IV1' refers to biological and physiological factors. 'IV2' refers to the symptom status of patients. 'IV3' refers to individual and environmental characteristics. IV1, IV2, and IV3 are independent variables.

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

H1') The change of IV1 and IV2 causes the change of the DV. (See Figure 3.2)

H2') The change of IV1 two causes the change of the DV. (See Figure 3.3) H3')

The change of IV2 causes the change of the DV. (See Figure 3.4)

H4') The change of IV1 causes the change of the DV. (See Figure 3.5) H5')

The change of IV3 causes the change of the DV. (See Figure 3.6)

As we have seen, there are almost only hypotheses that involve the bottom-up causation of 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, to Frank et al. (2004), the lower-level HRQL is the factor that causes the change of higher-level HRQL while the higher-level HRQL is always caused to be changed. In the other eight pieces of research, a similar situation was found.

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.

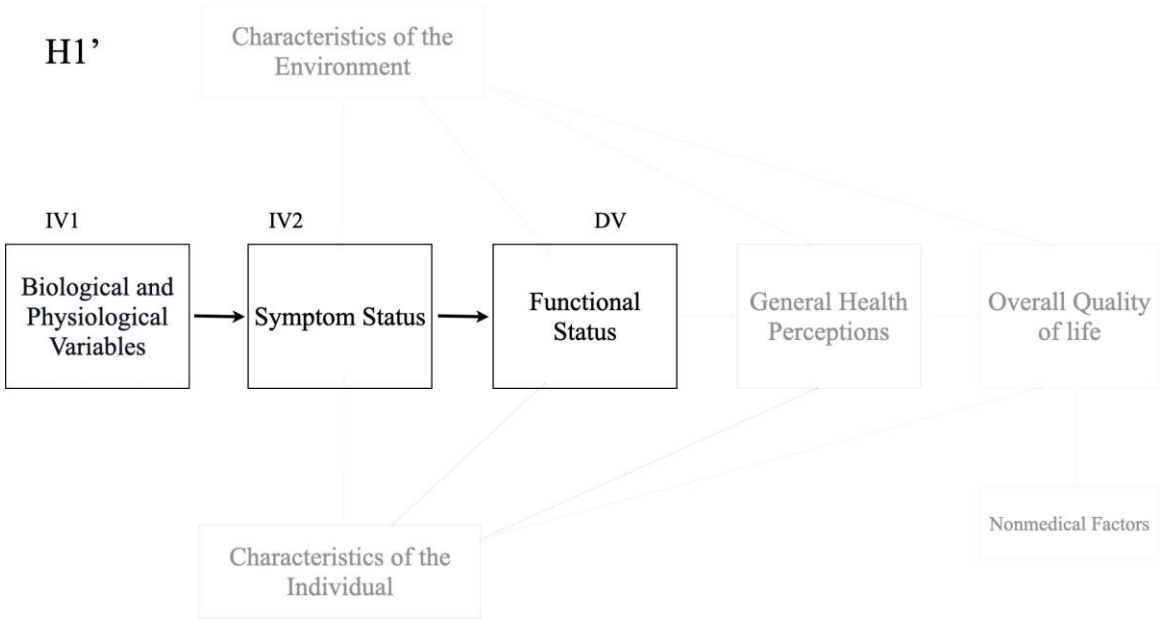

Figure 3.2: The First hypothesis in Frank et al. (2004). The description of arrows are omitted.

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

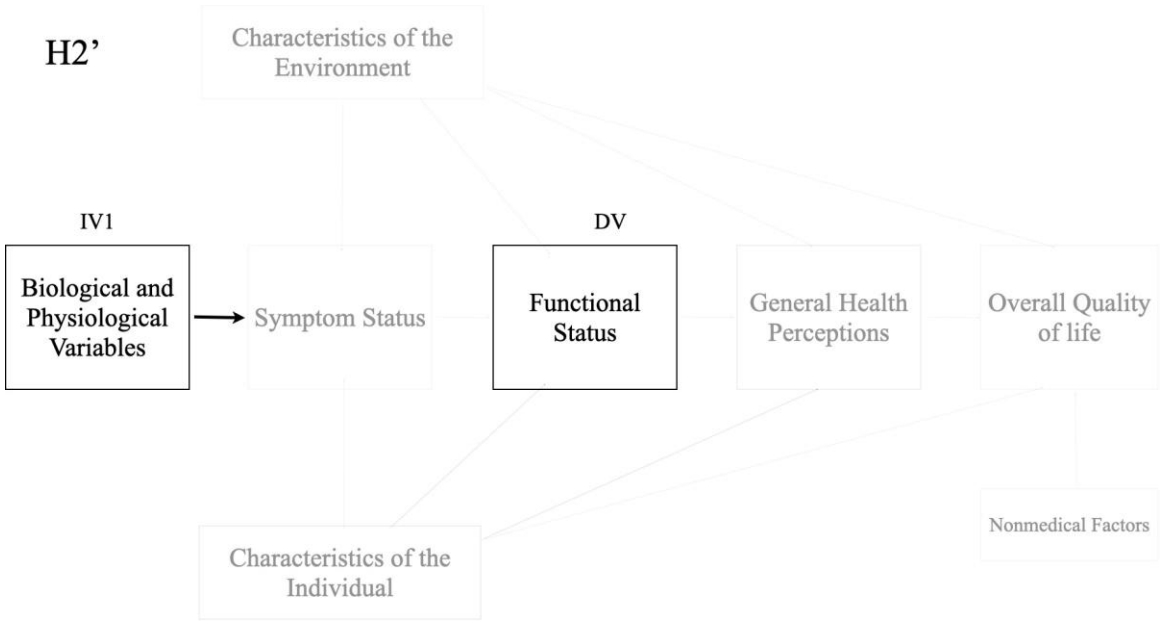

Figure 3.3: The second hypothesis in Frank et al. (2004). The description of arrows are omitted.

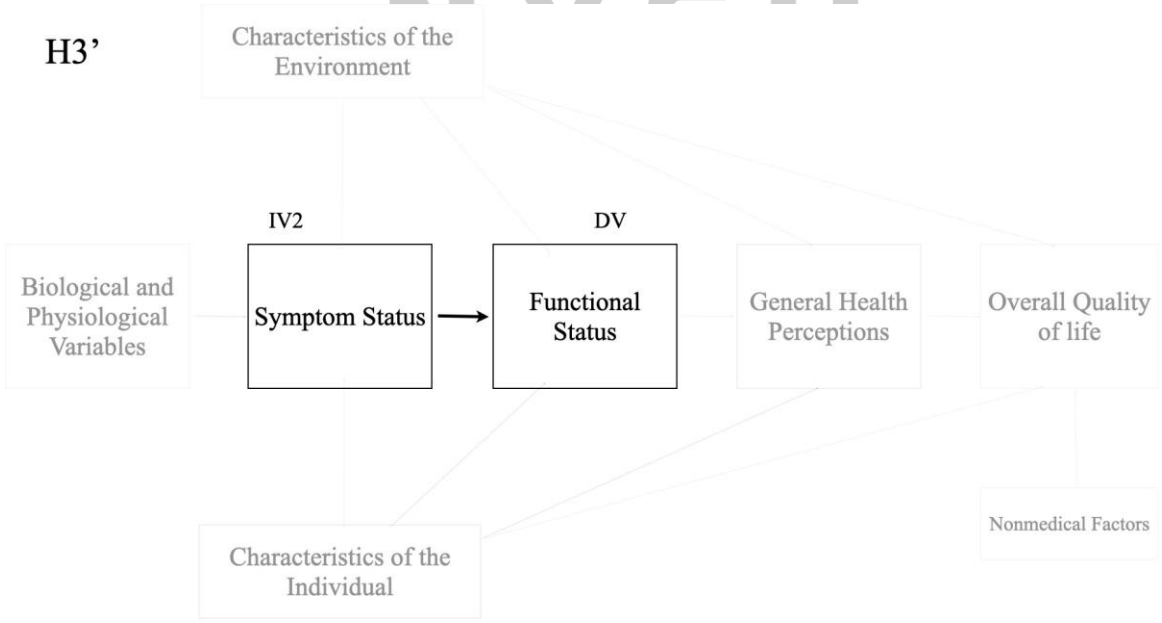

Figure 3.4: The third hypothesis in Frank et al. (2004). The description of arrows are omitted.

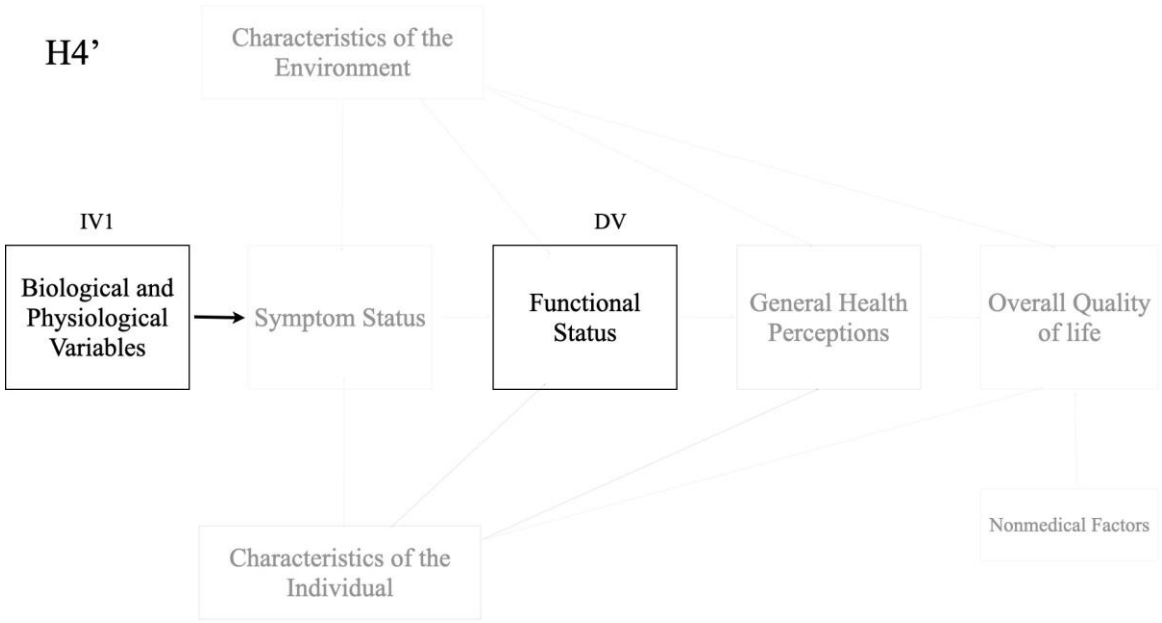

Figure 3.5: The Fourth hypothesis in Frank et al. (2004). The description of arrows are omitted.

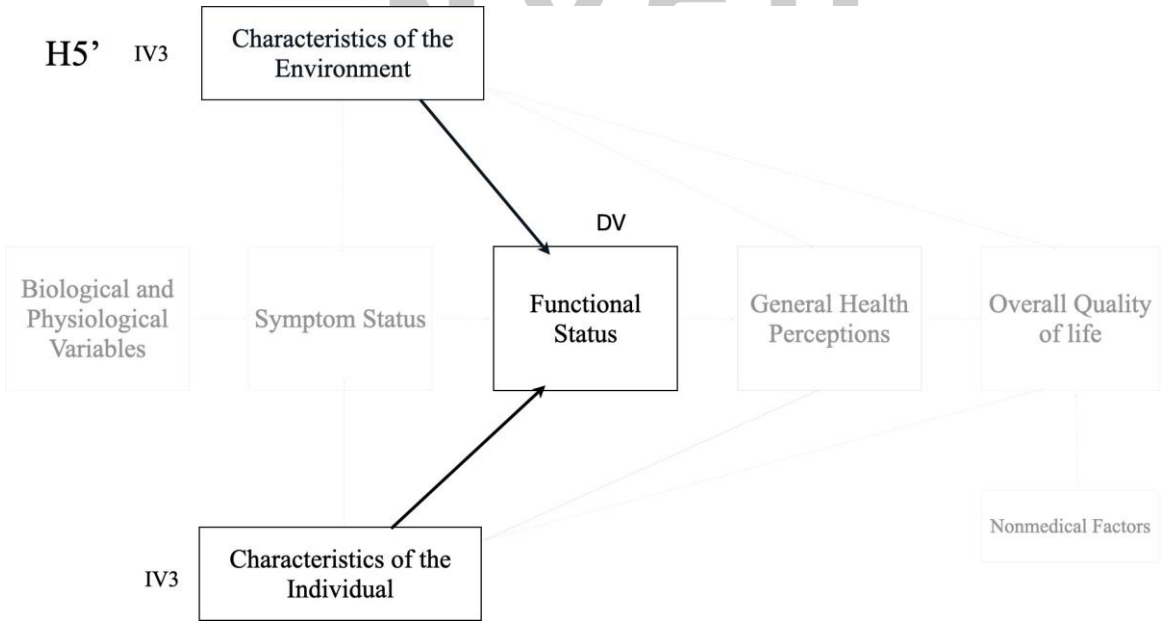

Figure 3.6: The Fifth hypothesis in Frank et al. (2004). The description of arrows are omitted.

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). In other words, the W &C model offer the concept of biological and physiological variables, and symptom status, that are used to predict HRQL of patients. Without the conceptual framework, how the two variables can be used? We can hardly say that Frank et al. (2004) would generate the same hypotheses without the W&C model. 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

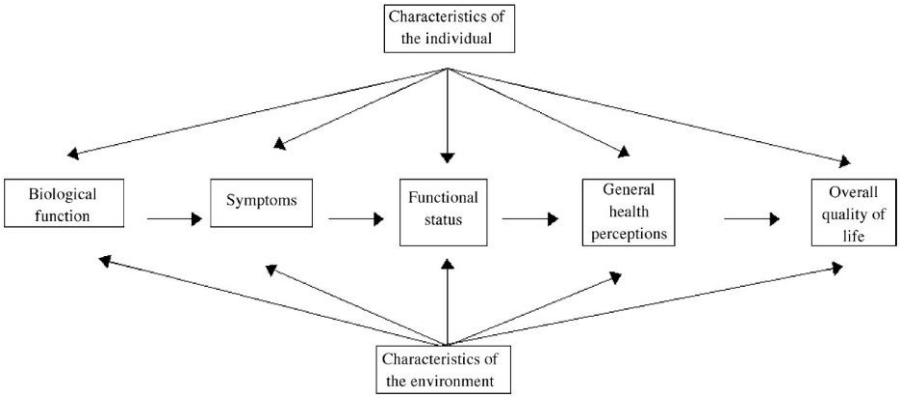

Figure 4.1: The HRQL theoretical model proposed by Ferrans et al. (2005). Reproduced from Ferrans et al. (2005).

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.

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 space. In this space no HRQL is more fundamental. 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 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

[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.

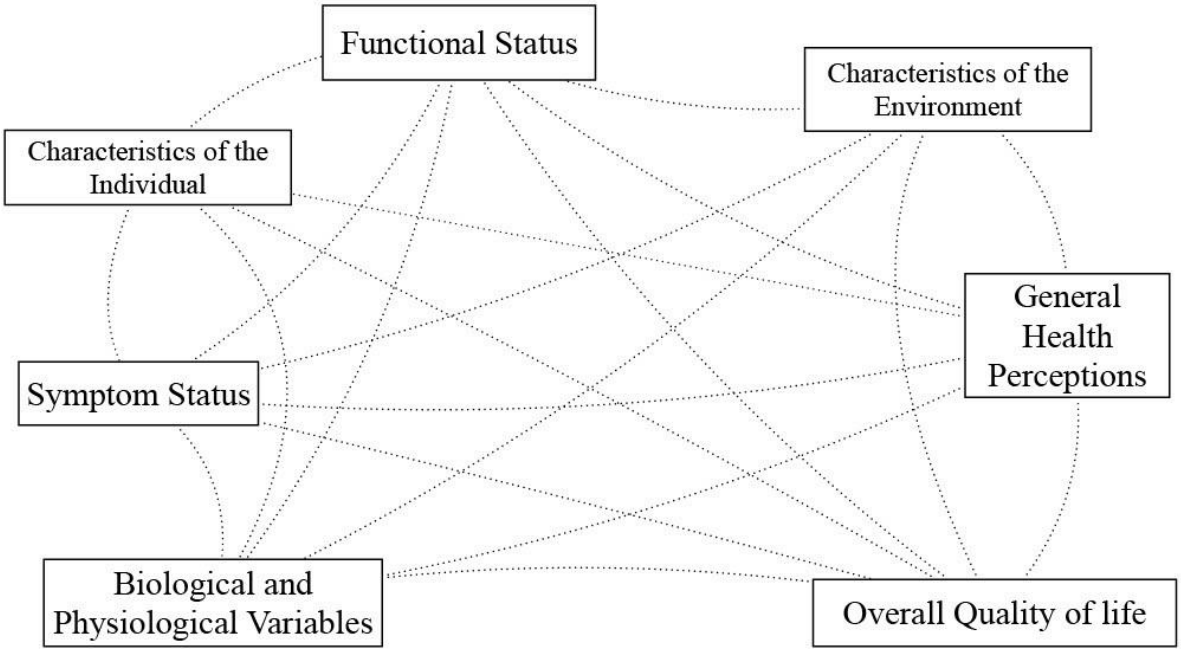

Figure 4.2: The revised HRQL theoretical model

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.

The revised model has some difference with Rocca and Anjum (2020). I have mentioned 'the other direction of causation' for several time, implied that I aimed to accommodate not only bottom-up and top-down causality. Does whether there is the other direction of causation matter? Or put in the other way, is it necessary to commit 'the other direction of causation' to avoid the causal bias? Rocca and Anjum (2020) introduced the top-down causality to medicine and healthcare in bio-psychosocial model. Hence their way to refuse the domination of bottom-up causality is to incorporate top-down causality. If so, then the question that how much top-down

causality do we want emerges. Is 50% enough? Or higher or lower? And why? I don't intend to deal with this problem here, since the problem is not the ratio but the qualitative aspect of the tools for practitioners: If the top-down causation or the other directions of causation are intended to be investigated, do the conceptual tools offer clear starting point? Either way does: I leave room for not just bottom-up or top-down causality, while Rocca and Anjum lead a force for investigating the top-down causality.

Using 'bottom-up' and 'top-down' is not the perfect expression in light of the revised model, but using them is acceptable: One might think that there is less investigation of top-down or the other direction of causation because they are hard to research. Probably clinical researchers have ideas about different directions of causation, yet the hypotheses testing bottom-up causation is more doable than testing top-down causation or the other directions. Therefore, hypothesis testing of bottom-up causation dominates clinical research. One way to refuse the domination is to force the investigation of top-down causalities like Rocca and Anjum did. In this sense, using 'bottom-up' and 'top-down' to discuss what I have discussed and elaborated on in the revised model is acceptable. However, there might be better expressions regarding talking about causation directions.

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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