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**Introducing the Causal Graph Approach to Psychopathology: An Illustration in
Patients with Obsessive-Compulsive Disorder**

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Abstract

Clinicians aim to discern causal relations among features of mental disorders. Yet observational data are insufficient to confirm these relations. In the present study, we describe a causal graph approach to illustrate how one can discern causal relations among features of obsessive-compulsive disorder (OCD) by applying the *Invariant Causal Prediction* (ICP) algorithm and the *Hidden Invariant Causal Prediction* (HICP) algorithm, supplemented by findings from a literature review. Applying these methods to symptom severity data from four time points (before, during [twice], and after behavioral treatment) we estimated a causal graph for OCD. The resulting causal graph reveals multiple cycles between aspects of OCD that may play a role in the maintenance of the disorder. We conclude that the proposed design has great potential and could benefit clinicians with some adjustments and extensions.

Introducing the Causal Graph Approach to Psychopathology: An Illustration in Patients with Obsessive-Compulsive Disorder

The network perspective is an emerging view that postulates that mental disorders form a system of interactive symptoms. Instead of a latent variable – the “underlying disorder” – causing symptoms that presumably reflect its presence, the symptoms and their interactions constitute the disorder, rendering the latent variable entirely redundant (Borsboom, 2017; Borsboom & Cramer, 2013). Figure 1 illustrates a causal graph: a network where the symptoms (here X , Y and Z .) are depicted as nodes (the circles), and the causal relations between symptoms are represented by an arrow (directed edge).

Clinical researchers aim to identify causal relations among symptoms. Unfortunately, their aspirations are often stymied by mere correlational data that can suggest, but never confirm, causal relations. Indeed, experimental manipulations designed to underwrite causal conclusions are frequently infeasible in clinical settings. However, new methods have emerged that enable such inferences (Kossakowski, Waldorp, & van der Maas, 2019). The purpose of our article is to introduce these methods by applying them to patients with obsessive-compulsive disorder (OCD).

In this study, we define a *causal relation* as a relation between two variables $X \rightarrow Y$ where, when we manipulate (or intervene on) variable X , we observe a change in variable Y (conditioned on all other variables; Kossakowski et al., 2019). This definition also means that, if we do not observe any changes in Y , no intervention has taken place on X . When it comes to the treatment of patients, it is important to know whether symptom X causes symptom Y , or perhaps vice versa. Figure 1 (left panel) illustrates a simple example where we have three variables, $X \rightarrow Y \rightarrow Z$. If the causal relations among these three variables are unknown, a therapist may first therapeutically target variable Z . However, as shown in this illustration, it may be more effective to target variables X and Y , as both of these have an (in)direct effect on variable Z .

Figure 1. Illustration of causal relations among three variables X , Y , and Z . The left panel depicts a Directed Acyclic Graph (DAG), whereas the right panel shows a Directed Cyclic Graph.

Many studies use *observational* data to estimate causal relations between variables. Observational data are data on which no interventions have taken place. However, observational data alone do not allow one to estimate causal relations accurately; one needs to manipulate variables to estimate true causal relations (Holland, 1986). Thus, we need *interventional* data upon which some manipulation or intervention has taken place. Intervention here indicates that some manipulation is performed compared to the observational data. Examples include starting treatment or reducing one's medication. By using both observational and interventional data, we could potentially estimate true causal relations between symptoms (Meinshausen et al., 2016).

In this study, we describe a causal graph approach in which we combine observational and interventional data to estimate a causal graph. This approach is illustrated with an application to data of patients diagnosed with OCD who were measured at four time points: before, twice during, and after treatment. OCD is characterized by intrusive, persistent thoughts, urges or images (obsessions), and repetitive behaviors or mental acts (compulsions) performed to relieve the distress that accompanies the obsessions (American Psychiatric Association [APA], 2013). US national and global life-time prevalence rates for OCD range from between 1.6% to 2.3% (e.g., Fontenelle, Mendlowicz, & Versiani, 2006; Kessler et al., 2005; Ruscio, Stein, Chiu, & Kessler, 2010). Two network studies on OCD estimated

Directed Acyclic Graphs (DAGs) involving features of OCD and symptoms of depression (Jones, Mair, Riemann, Mugno, & McNally, 2018; McNally, Mair, Mugno, & Riemann, 2017). A DAG illustrates potential predictive causal relations among symptoms that together depict a causal system (Pearl & Mackenzie, 2018), but only if two stringent assumptions are met. The causal graph must contain all important variables, and there cannot be any cycles among the nodes. A cycle is shown in Figure 1 (right panel), where variable Z is the cause of variable X, thereby creating a cycle.

Ideally, to estimate causal relations, we would want to manipulate one symptom at a time, and observe its effects on other symptoms. This approach would mimic an approach in gene biology whereby researchers “knock-out” or silence a specific gene and observe the subsequent effects (Pinna, Soranzo, & de la Fuente, 2010; Serra, Villani, & Semeria, 2004). Using a “slim finger,” geneticists can deactivate a gene while leaving all other genes untouched. Unfortunately, clinical psychologists are saddled with “fat fingers” that render it difficult to deactivate a single symptom without simultaneously affecting other symptoms. For example, it would be nearly impossible to administer a benzodiazepine for insomnia without also reducing anxiety at the same time. Ironically, targeting one symptom (e.g., insomnia) will not only affect other symptoms in *serial* fashion (e.g., less fatigue the following day), but will also produce effects on additional symptoms in *parallel* fashion (e.g., anxiety), thereby complicating causal analysis. Indeed, algorithms designed for gene knock-out experiments may not work as accurately when applied to psychological data (Kossakowski et al., 2019).

In the causal graph approach, we applied the *Invariant Causal Prediction* (ICP) algorithm (Meinshausen et al., 2016) and the *Hidden Invariant Causal Prediction* (HICP) algorithm (Peters, Buhlmann, & Meinshausen, 2016; Peters, Janzing, & Schölkopf, 2017). These algorithms represent a significant advance over DAGs as the daunting assumptions of the latter are difficult to meet in clinical science. The ICP-algorithm can handle interventional data where multiple symptoms are simultaneously manipulated. The HICP-algorithm complements the ICP-algorithm because it can accommodate *hidden variables* – unmeasured variables that may affect the measured variables nonetheless (Peters et al., 2016, 2017). Both algorithms have shown great potential for psychological applications (Kossakowski et al., 2019). Lastly, following Meinshausen et al.'s (2016) approach, we also searched the literature for evidence of causal relations between features of OCD. We combined results from the ICP-algorithm, the HICP-algorithm and the literature review into a single causal graph that may give an overview of the causal structure of OCD.

Methods

Participants

We used archival clinical data from patients with a primary OCD diagnosis. A total of 3474 patients entered the study at one of the ten locations of the Rogers Behavioral Health centers across the United States treated between June 2012 and June 2018. Patients received treatment either in an intensive outpatient ($n = 1320$), partial hospitalization ($n = 1247$), or in a residential unit ($n = 906$). The treatment setting for one patient was unknown. The primary intervention was exposure and response prevention therapy. We included only those patients who completed the relevant questionnaires prior to treatment, at the first and second progress assessments, and the post-treatment assessment ($t = 4$ time points). We ended up with 903 patients that were included for all analyses. Patients gave their written informed consent to use de-identified data for research purposes. Of this sample, 282 patients received treatment in an intensive outpatient unit, 371 patients received treatment in a partial hospitalization unit, and 250 patients received treatment in a residential unit. One patient switched from an intensive outpatient unit to a partial hospitalization unit after the initial measurement. The average number of days between admission and discharge was 56.39 ($SD = 20.29$ days). The average time between measurements ranged from 15.81 to 23.25 days, with a standard deviation ranging from 7.43 to 17.67 days. The sample consisted of 478 (52.93%) females and 424 (46.95%) males, whose age ranged from 18 to 76 ($M = 29.69$, $SD = 11.42$). The gender of one participant was unknown. Based on the recommendations presented by Mataix-Cols et al. (2016), who argue that a treatment response is shown by a reduction in Y-BOCS score by at least 35% compared to their score at baseline, 538 patients in our sample experienced a clinically meaningful reduction in symptoms.

Yale-Brown Obsessive-Compulsive Scale Self-Report

Patients completed a test battery that contained, among other questionnaires, the *Yale-Brown Obsessive-Compulsive Scale Self-Report* (Y-BOCS-SR; Steketee, Frost, & Bogart, 1996), the self-report version of the Y-BOCS (Goodman, Price, Rasmussen, Mazure, Fleischmann, et al., 1989). Patients completed the Y-BOCS-SR at four time points before, during, and after treatment. The Y-BOCS-SR is a 10-item questionnaire that evaluates features of OCD without taking the *content* of a patient's obsessions and compulsions into account (e.g., harming versus contamination obsessions; checking versus washing compulsions). The Y-BOCS-SR has a symmetric structure: the questionnaire evaluates five

aspects of obsessions and of compulsions, respectively. Table 1 gives a more specific overview of the individual items, including the means and standard deviations of each item per measurement. All items were measured on a five-point Likert scale, ranging from 0 (no symptoms) to 4 (extreme). The Y-BOCS-SR shows excellent psychometric properties (e.g., Goodman, Price, Rasmussen, Mazure, Delgado, et al., 1989; Goodman, Price, Rasmussen, Mazure, Fleischmann, et al., 1989; Steketee et al., 1996).

Table 1. The Yale-Brown Obsessive Compulsive Scale Self-Report (Y-BOCS-SR) with their assigned item labels and mean (SD) scores per time point.

Item	Item label	Mean (SD) Baseline	Mean (SD) Progress1	Mean (SD) Progress2	Mean (SD) Post
Time occupied by obsessive thoughts	O-Time	2.66 (1.15)	2.34 (1.04)	2.16 (1.05)	1.76 (0.98)
Interference due to obsessive thoughts	O-Int	2.39 (1.06)	1.99 (0.93)	1.77 (0.95)	1.41 (0.89)
Distress associated with obsessive thoughts	O-Distr	2.55 (1.02)	2.12 (0.89)	1.97 (0.91)	1.64 (0.87)
Resistance against obsessions	O-Res	1.72 (0.99)	1.42 (0.85)	1.27 (0.85)	1.01 (0.88)
Degree of control over obsessive thoughts	O-Cont	2.32 (0.96)	1.91 (0.83)	1.74 (0.81)	1.42 (0.79)
Time spent performing compulsive behaviors	C-Time	2.17 (1.16)	1.81 (1.03)	1.67 (0.99)	1.41 (0.95)
Interference due to compulsive behaviors	C-Int	2.11 (1.16)	1.74 (1.04)	1.54 (0.97)	1.28 (0.95)
Distress associated with compulsive behavior	C-Distr	2.36 (1.06)	2.05 (0.93)	1.91 (0.93)	1.56 (0.90)
Resistance against compulsions	C-Res	1.89 (1.06)	1.40 (0.84)	1.23 (0.85)	0.95 (0.82)

Degree of control over compulsive behavior	C-Cont	2.16 (1.00)	1.74 (0.86)	1.56 (0.83)	1.28 (0.79)
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Causal Graph Approach

The proposed causal graph approach combines evidence for specific directed edges from three different sources: the ICP-algorithm (Meinshausen et al., 2016), the HICP-algorithm (Peters et al., 2017), and a literature study.

Invariant Causal Prediction

The ICP-algorithm (Meinshausen et al., 2016; Peters et al., 2016) is designed to handle the combination of observational and interventional data. The basic idea is that the conditional distribution of a single node (called the *target node*), given its direct causes, does not change if some manipulation takes place on variables other than the direct causes of a target node. Hence, for each node, the ICP-algorithm ascertains the subset of remaining nodes that cause the target node. The algorithm starts by selecting one node, which is the *target node*. The target node is then regressed on each possible subset of remaining nodes, ranging from an empty subset, to all remaining nodes in the graph. For each of these regressions, we obtain the residual distribution. This is then split according to an *instrumental variable*. This variable indicates which score (i.e., residual) belongs to which measurement time point. The algorithm then compares the residuals between every pair of measurements (4 in our illustration: before, twice during, and after treatment). If the residuals between a pair of measurements are deemed equal, we conclude that the causal relation between the subset of nodes and the target node is “invariant.” After all subsets are examined, we draw directed edges from the intersection of all subsets to the target node. If, for a given subset of nodes, the residuals are similar across all measurements, then conditional on the direct causes of the target node, any manipulation will not change the distribution of the residuals of the target node when one conditions on the direct causes. This process is then repeated for all nodes in the graph. For a more detailed description of how the ICP-algorithm performs in a simulation study, see Kossakowski et al., (2019) and Meinshausen et al. (2016).

Hidden Invariant Causal Prediction

When we run the ICP-algorithm, we assume that we have measured all possible causes of every single node. However, it is very difficult to satisfy this assumption, especially in psychopathology. Many aspects or events can affect features of OCD that are not captured by the Y-BOCS-SR. For example, research has shown that the catastrophic misinterpretation of obsessive thoughts (Rachman, 1997) can influence OCD symptoms. Also, OCD tends to co-occur with other mental disorders such as anxiety or mood disorders (see Jones et al., 2018; McNally et al., 2017; Ruscio et al., 2010; Ruzzano, Borsboom, & Geurts, 2015). Therefore, we ran the HICP-algorithm to account for the influence of *hidden variables*: variables that are not measured, but that may affect those we did measure (Meinshausen et al., 2016; Peters et al., 2017).

A hidden variable that affects both the cause and the target node inflates the relation between the two variables if the hidden variable is not accounted for. When hidden variables affect variables of interest, the ICP-algorithm can no longer estimate the causal relation between two variables accurately. Accordingly, to estimate the true causal relation between two variables, the HICP-algorithm employs an *instrumental variable*. An instrumental variable influences only the cause, but not the target node. It is the same variable that we used in the ICP-algorithm to split the data according to the different time points in the dataset. An instrumental variable splits the data into four parts that correspond to the four assessments that we have for our data. By comparing every pair of time points, the HICP-algorithm partials out the effect of the hidden variable. For more information on the HICP-algorithm and how it performs in a simulation study, see Kossakowski et al. (2019).

Literature Search

We aimed to complete the causal graph that results from the ICP and HICP-algorithm by means of a literature study. Using Google Scholar and Web of Science, we searched for articles on OCD. Within these studies, we looked for sentences that signify a causal relation between specific aspects of OCD. For example, Marker, Calamari, Woodard, and Riemann (2006, p.390) state that “Obsessive-compulsive disorder (OCD) is a common anxiety disorder characterized by intrusive thoughts that are difficult to dismiss and that increase anxiety.” A sentence such as this expresses a causal relation from *O-Time* (time occupied by obsessive thoughts) to the subsequent *O-Distr* (distress associated with obsessive thoughts). An independent researcher unaware of our results double-checked our findings as a safeguard against any possible subjectivity and confirmation bias.

Results

Literature Study

The two researchers – the first two authors of this paper – found a total of 74 causal relations in 15 articles and two books. Researcher R1, who also searched for the literature, found 53 causal relations, and researcher R2 56. Of these causal relations, initially 21 were found by both researchers in the same materials. The initial lack of overlap between the two researchers was due to a different interpretation of the resistance items (*O-Res* and *C-Res*). After deliberation between the two researchers, the overlap increased to 35 causal relations, of which nine represent unique causal relations. We decided to include only those causal relations found by both researchers. Supplementary Table S1 gives an overview of all the edges that are in the causal graph with the corresponding source.

Causal Graph

Figure 2 presents the causal graph, in which both show the split results from the algorithms and the literature review (Figures 2a and 2b), and a causal graph (Figure 2c), where we combined the results from the ICP, the HICP-algorithm, and the literature review. Supplementary Table S1 gives an complete overview of the sources justifying inclusion of edges in Figure 2. The first characteristic that stands out in this causal graph is the lack of overlap between the different sources. Out of the 14 edges that were detected, three edges were detected by both the ICP-algorithm and the HICP-algorithm, and only two edges were detected by either an algorithm and the literature review. At the same time, many edges appeared at least two studies or sources; only five edges had only one source (algorithm or literature).

Another interesting aspect of this causal graph is that it depicts four cycles. The first cycle (detected by both the ICP and the HICP algorithms) exists between *C-Distr* and *C-Cont*, which indicates that, when for example the score on *C-Distr* (distress associated with compulsive behavior) changes, this causes the score on *C-Cont* (degree of control over compulsive behavior) to change as well, and vice versa. This means that, on average, when patients score higher on *C-Cont*, and thus have a lower level of control over their compulsions, their score on *C-Distr* increases as well, and they on average experience more distress associated with their compulsions.

The second cycle in Figure 2a and 2c is between *C-Time* and *C-Int*. This cycle, which is only detected by the HICP-algorithm, may indicate that, when patients' score on *C-Time* (time spent on compulsive behaviors) changes, this causes the score on *C-Int* (interference

due to compulsive behaviors) to change as well, and vice versa. This means that, on average, when patients score higher on *C-Time*, and thus spend more time performing compulsive behaviors, their score on *C-Int* changes as well, and they on average experience more interference in their daily life due to their compulsive behaviors.

The third cycle was between *O-Distr* and *C-Time* (Figure 2b and c). This cycle appeared only in the literature and suggests that when patients' score on *O-Distr* (distress associated with obsessive thoughts) changes, this causes the score on *C-Time* (time spent on compulsive behaviors) to change as well, and vice versa. This means that, on average, when patients score higher on *O-Distr*, and thus on average experience more distress due to their obsessive thoughts, their score on *C-Time* increases as well, and they spend more time performing compulsive behaviors.

The fourth and last cycle was between *C-Time* and *O-Time*, and also only appeared in the literature. This cycle indicate that, as patients' score on *O-Time* (time spent on obsessive thoughts) changes, their score on *C-Time* (time spent on compulsive behaviors) changes as well, and vice versa. Thus, spending more time on obsessive thoughts results in spending more time on compulsive behaviors, which in turn leads to spending more time on obsessive thoughts.

Figure 2. Causal graph of the Y-BOCS-SR items, detected by the ICP and HICP algorithms (a), found in the literature (b), and combined (c). Item labels are described in Table 1. White nodes signify aspects of obsession, whereas gray nodes signify aspects of compulsions. Solid edges are those detected by either of two algorithms *and* in the literature. Dashed edges are those found with either of two algorithms, or in the literature. The edge thickness and color saturation shows how often an edge is detected, the thicker and more saturated an edge, the more often it was detected.

Interestingly, Figure 2 shows that the item *O-Time* is the cause of several other nodes and only received causal influence from the item *C-Time*. This could imply that the amount of time spent obsessing is the key driver of the causal system depicted in the graph, at least for those individuals already diagnosed with OCD. This seems to confirm the (dominant) view that OCD starts with obsessive thoughts, although not everyone agrees with this (Gillan & Sahakian, 2015). Lastly, two clinical features are disconnected from the rest of the causal graph, *O-Res* and *C-Res*. It is possible that this symptom is disconnected because it is part of the treatment (exposure and response prevention) that was given to the patients that were part of this study.

Notably, only three causal relations connect the obsessive items with the compulsive ones, $O-Time \leftrightarrow C-Time$, $O-Distr \leftrightarrow C-Time$ (both found in the literature) and the causal relation $O-Distr \rightarrow C-Int$ (found with the ICP-algorithm), which in turn is also causally related to *C-Time*. This implies that there are only a few pathways whereby obsessive thoughts can lead to compulsions in patients, either directly or via some alternative path.

Discussion

In this exploratory study we introduced a causal graph approach and applied it to data of patients with OCD who completed the Y-BOCS-SR four times before, (twice) during, and after treatment. Using two algorithms, we estimated a causal graph for OCD by combining observational and interventional data. The addition of interventional data enables the estimation of causal relations that are hard to detect with only observational data. The resultant causal graph generated by the algorithms was complemented with a literature study. Results exposed several cyclic patterns between features of OCD. These may signify subsystems that maintain (parts of) the disorder throughout therapy.

The ICP and HICP-algorithms did not estimate many causal relations between the items of the Y-BOCS-SR. The ICP-algorithm estimated six causal relations, and the HICP-algorithm estimated five causal relations, three of which were estimated by both algorithms.

In a graph where 45 different edges are possible, it is unclear whether the few causal relations detected by these algorithms exhaust all of them or whether limited statistical power may have rendered others difficult to detect. Indeed, Kossakowski et al. (2019) showed that the ICP-algorithm can be quite conservative and will only use the intersection of all the subsets that are investigated. This means that edges will only be estimated when a specific edge appears in all the subsets that have significant causal relations with the target variable (i.e., the item that is the recipient of the causal influence). The conservative character of the ICP-algorithm is also shown by its high accuracy when the intervention is strong with a small variance, which is an aspect of interventional data that is difficult to ascertain. At the same time, Kossakowski and colleagues showed that the HICP-algorithm has a high sensitivity, but only a moderate to high specificity depending on the number of edges in the graph and the strength of the intervention. The lack of variance within the items could also have caused the small number of causal relations to be estimated. However, as shown in Table 1, the standard deviations of the items per time point are high enough to suggest otherwise.

The causal graph that shows the combined result of the ICP and HICP-algorithm visualized a discrepancy between these two algorithms and the results of the literature study. Only two causal relations that were detected by either of the two algorithms were confirmed with the literature study. It is quite possible that the causal relations estimated with the algorithms exist in the literature, but that we have not found the right studies that show evidence for these relations. Even though many articles were found, not all articles explicitly stated a causal relation between any two features of OCD, other than its two global, defining symptoms: obsessions and compulsions. A search on Web of Knowledge uncovered more than 5500 articles exist with the term “obsessive-compulsive disorder” in their title, so 17 different sources may not be representative of the existing literature on OCD. Another disadvantage of the current literature study is that the used sources did not experimentally test if one feature of OCD (e.g., time spent on obsessive thoughts) had a causal effect on another (e.g., distress associated with compulsions).

The ICP and HICP-algorithm state that a causal relation between two variables can only be *invariant* when the residual distributions between time points are equal. This means that the causal relations found by the ICP and the HICP-algorithm reflect those causal

relations that exist throughout therapy. Causal relations that change due to therapy are not included, as these relations cannot be picked up by either the ICP-algorithm or the HICP-algorithm. Although it is interesting to explore the causal relations that are not affected by therapy, it can also be interesting to study those causal relations that vanish as a result of therapy. Furthermore, in this study we examined patients who underwent treatment. We did not have access to a control or waitlist condition for which we could run the same analyses and compare the results. In future studies, adding a control condition and comparing results could illuminate differences attributable to therapy. Another option would be to use therapy as a dummy variable (when the data consists of an experimental and control condition) and estimate the causal graph with that dummy variable, similar to the approach that Blanken, van der Zweerde, van Straten, van Someren, and Borsboom (2019) used.

To our knowledge, this is the first study that estimates a causal graph for OCD using both observational and interventional data. By combining results from different algorithms and sources, we may uncover causal relations between features that otherwise are left unknown. The result of this causal graph approach may potentially be used to set up intervention strategies in a therapy setting. Even though we have to investigate to what extent this causal graph approach is useful and meaningful for clinical practice, we do believe that the causal graph approach has a great potential for illuminating the causal structure of psychopathology.

Author Contribution

J.J.K., R.J.M., L.J.W., and H.L.J.M. jointly generated the idea for the study. J.J.K. programmed the study. B.C.R. was responsible for the data collection. J.J.K and L.J.B.O performed the literature study. J.J.K analyzed the data under the supervision of R.J.M, L.J.W and H.L.J.M. J.J.K. drafted the manuscript and L.J.B.O., R.J.M., B.C.R., L.J.W., and H.L.J.M provided critical revisions. All authors approved the final version of the paper for submission.

Conflicts of Interest

The authors declare that there were no conflicts of interest with respect to the authorship or publication of this article.

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465 **Supplementary Table S1.** Origin of all edges present in Figure 2.

Edge	Algorithm	Literature
O-Time – O-Int		Goodman, Rudorfer, & Maser (2000, chapter 1, p. 33)
O-Time – O-Distr	ICP	<ul style="list-style-type: none"> - Goodman, Rudorfer & Maser (2000, chapter 1, p. 33) - Goodman, Rudorfer & Maser (2000, chapter 7, p. 239) - Goodman, Rudorfer & Maser (2000, chapter 11, p. 395) - Okasha (2002, chapter 1, p. 2) - Jacoby, Leonard, Riemann, & Abramowitz (2016, p. 179) - Janeck, Calamari, Riemann & Heffelfinger (2003, p. 182) - Blakey, Abramowitz, Reuman, Leonard & Riemann (2017, p. 113) - Marker, Calamari, Woodard & Riemann (2006, p. 390)
O-Time – O-Cont		- Janeck, Calamari, Riemann & Heffelfinger (2003, p. 182)
O-Time – C-Time		<ul style="list-style-type: none"> - Goodman, Rudorfer & Maser (2000, chapter 6, p. 223) - Goodman, Rudorfer & Maser (2000, chapter 11, p. 404)
O-Int – O-Distr	ICP + HICP	
O-Distr – C-Time		<ul style="list-style-type: none"> - Najmi, Riemann, & Wegner (2009, p. 494) - Manos, Cahill, Wetterneck, Conelea, Ross & Riemann (2010, p. 700)
O-Distr – C-Int	ICP	
O-Cont – O-Int	ICP	
C-Time – O-Time		- Goodman, Rudorfer & Maser (2000, chapter 6, p.

		223)
		- Goodman, Rudorfer & Maser (2000, chapter 6, p. 229)
		- Goodman, Rudorfer & Maser (2000, chapter 7, p. 241)
		- Goodman, Rudorfer & Maser (2000, chapter 11, p. 393)
		- Goodman, Rudorfer & Maser (2000, chapter 11, p. 399)
		- Najmi, Riemann, & Wegner (2009, p. 495)
		- Hezel & McNally (2016, p. 222)
		- Hezel & McNally (2016, p. 223)
		- Hezel & McNally (2016, p. 223)
		- McNally & Ricciardi (1996, p. 17)
		- Tolin, Abramowitz, Przeworski, & Foa (2002, p. 1256)
C-Time – O-Distr		- Goodman, Rudorfer & Maser (2000, chapter 7, p. 241)
		- Goodman, Rudorfer & Maser (2000, chapter 11, p. 393)
		- Goodman, Rudorfer & Maser (2000, chapter 11, p. 395)
		- Goodman, Rudorfer & Maser (2000, chapter 11, p. 399)
		- Najmi, Riemann & Wegner (2009, p. 495)
C-Time – C-Int	HICP	Goodman, Rudorfer & Maser (2000, chapter 1, p. 33)
C-Time – C-Distr		- Goodman, Rudorfer & Maser (2000, chapter 1, p. 33)
		- Goodman, Rudorfer & Maser (2000, chapter 7, p. 239)
		- Luchian, McNally, & Hooley (2007, p. 1657)
		- Okasha (2002, chapter 1, p. 3)

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C-Int – C-Time	HICP
C-Distr – C-Cont	ICP + HICP
C-Cont – C-Distr	ICP + HICP

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