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Original Paper

Misconceptions Created by Tulodziecki's Revisionist Account of Semmelweis's Theory and Reasoning in the Philosophy of Science Literature

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Abstract

Semmelweis's work on the nature and cause of childbed fever has been used as a 'paradigm case' by philosophers of science "to illustrate aspects of the confirmation of theory by data" for more than fifty years (Scholl & Räz 2016). However, in 2013, Dana Tulodziecki challenged this paradigmatic view, and argued, based on a reconstruction of Semmelweis's work that differed from "the standard story as it is found in the extant philosophical literature on Semmelweis", that Semmelweis was "not the excellent reasoner he has been supposed to be." Philosophers of science have accepted Tulodziecki's reconstruction of Semmelweis work at face value as valid, and have already used it to question the philosophical theses Semmelweis work has been used to illustrate. The purpose of this article is to cut short this revisionist trend by demonstrating that, based on the Semmelweis's own account of his theory and reasoning, and on other contemporaneous publications, Tuloziecki's account of Semmelweis's work is in every material respect incorrect and historically untenable.

Keywords

Semmelweis, Tulodziecki, cause of childbed fever

1. Introduction

The Hungarian obstetrician, Ignaz Philipp Semmelweis (1818-1865), is famous for discovering how parturient women developed a disease called childbed fever (CBF) (Note 1), what the nature of the disease was, and how the disease, which was the leading cause of maternal mortality at the time, could be prevented. Philosophers of science have used Semmelweis's work so frequently "to illustrate, appraise and compare methodological proposals" that in 2013, Scholl (p. 67) concluded that it was "no

exaggeration to say that the Semmelweis case has become a paradigm of scientific discovery and confirmation within the philosophy of science." However, in the same year, Dana Tulodziecki published an entirely different account of Semmelweis's discovery and reasoning. Her purpose in doing so was to refute Gillies's suggestion that Semmelweis's theory was rejected by his contemporaries because the requisite Kuhnian paradigm shift necessary to accept his theory had to await the discovery of bacterial causes of human disease, which had not yet occurred. (Gillies, 2005).

Tulodziecki argued that

> the reason Semmelweis's views were rejected is that he simply did not provide particularly strong arguments in their favor, and that he failed to address eminently reasonable objections being put to him by his contemporaries. (Tulodziecki, 2013, p. 1066)

And she envisaged that her revisionist account of Semmelweis's discovery would undermine the philosophical theses that Semmelweis's work had been used to illustrate:

> Ultimately, it will be important to show in exactly what ways the revised story of Semmelweis undermines the various philosophical accounts. This is a substantial undertaking . . . so I will restrict myself to showing that the standard view is, indeed, mistaken. (Tulodzieki, 2013, p. 1066, n.2)

Philosophers of science have accepted Tulodziecki's revisionist account of Semmelweis's theory and reasoning at face value as valid, and have already begun the undertaking that Tulodziecki envisaged her account would necessitate:

> This [Lipton's description] is not an accurate description of Semmelweis's research. Through a close historical examination of his research, Dana Tulodziecki (2013) convincingly argues that Semmelweis was not as perfect a reasoner as he is depicted by many philosophers of science—including Lipton—to be. (Mohammadian, 2016, p. 4214 n.10; *accord* Scholl & Raz, 2016, p. 11: "Tulodziecki . . . has recently argued that the discussion of Semmelweis proceeds from the false assumption that Semmelweis was an excellent reasoner. She discusses a number of flaws in Semmelweis's arguments which indicate that the case is not, after all a representative instance of successful scientific reasoning.")

The purpose of this article is to demonstrate that Tulodziecki's revisionist account of Semmelweis work is in every material respect incorrect and historically untenable, and thereby prevent her account of Semmelweis's work from being perpetuated in the philosophy of science literature.

2. Background

The elements of the Semmelweis story have been recounted many times and are well known. The lying-in section of the Vienna General Hospital (*Allgemeines Krankenhaus*-AKH) where Semmelweis worked had two maternity divisions (Divisions I & II). Medical students were taught exclusively in Division I, and student midwives exclusively in Division II, and only medical students were involved

with autopsies. Women were admitted to the two divisions from the same pool of non-fee paying patients on essentially alternate days—on Mondays, Wednesdays and Fridays to Division II, and on Tuesdays, Thursdays and the weekend to Division I—yet the MMR from CBF in Division I was three to nine times higher than in Division II. In some years more than five hundred women died of CBF in Division I causing so much public alarm that pregnant women pleaded not to be admitted to the first division or contrived to deliver outside the hospital (the so-called 'street births'). This was the problem that confronted Semmelweis when he was appointed First Assistant in Division I on July 1, 1846, his twenty-eighth birthday—a problem that several investigating committee appointed by the Ministry of Education to address had been unable to solve. (Semmelweis, p. 387)

What is less well known is that Semmelweis "belonged to a group of students who gathered around Rokitansky and his assistant Jakob Kolletschka (1803-1847) in a particularly intimate circle," (Note 2) and that Semmelweis spent much of the two years after graduating as a physician in April, 1844, and before his appointment as First Assistant, performing autopsies in Rokitansky's morgue on mothers who had died of CBF and on their babies if they too had died, which was often the case. (Note 3) In the course of this work, Semmelweis made an important empirical observation from which he derived the two inductive generalizations that were the major premises of his deductive inferences about the nature and cause of CBF. He noted that the pathological changes at autopsy in newborns who died after birth, and who had been born to mothers who had died of CBF, were the same as those in their mothers, except for the internal genitalia. (Semmelweis, 1941, p. 392) Semmelweis reasoned that if the pathological findings were the same the diseases producing those findings must also have been the same, otherwise it would render anatomic pathology meaningless. (Note 4) He also assumed that if the diseases were the same their causes must also be the same (Note 5):

> The anatomical findings in the cadavers of such newborns were, with the exception of the genital organs, identical with findings in the dead bodies of puerperae who succumbed to puerperal fever. To recognize the changes in the bodies of the puerperae and not to recognize the identical results in the bodies of the newborn, invalidates pathological anatomy.
>
> If it is one and the same disease from which the puerperae and the newborn die, then there must be the same etiology for the newborn which is admitted as applicable to the mother.' (Semmelweis, 1941, p. 381).

2.1 The Empirical Bases and Logic of Semmelweis's Inferences

Semmelweis spent a total of 28 months as First Assistant in Division I in two separate stints separated by five month. The first period lasted less than 4 months as Semmelweis's predecessor, Franz Breit, was granted an extension of his assistantship, and Semmelweis had to step down as First Assistant on October 20, 1846. Semmelweis resumed his position as First Assistant on March 20, 1847 after Breit was offered a professorship in Tübingen.

2.1.1 Investigations during the First Period of Semmelweis's Assistantship

During the first period, Semmelweis investigated whether the accepted causes of CBF could explain the difference in the incidence of CBF between the two maternity divisions. From the fact that women were admitted to the two maternity divisions on essentially alternated days, Semmelweis concluded that the cause of the disproportionately high MMR from CBF in Division I must be endemic, and not epidemic as was generally believed:

> Otherwise one will be forced to the absurd assumption that lethal epidemic influences must be subject to twenty-four-hour remissions and exacerbations in their pernicious activity, and that the remissions, through successive years, just coincide with the admission days for the Second Clinic, while the exacerbations over a number of years set in exactly at the time admissions are made to the First Division. (Semmelweis, 1941, p. 358)

Semmelweis then examined the potential role of each putative factor by determining whether or not their practical implications were realized. For example, he reasoned that if overcrowding caused CBF as generally believed, the months in which the MMR from CBF was the greatest should have the greatest number of deliveries, but found that this was not the case, (Semmelweis, pp. 363-375), and so on with every putative cause that Semmelweis investigated. (Ibid., pp. 379-380, p. 390) When the pragmatic consequences of a putative cause could not be tested—e.g. that pregnancy itself caused CBF—Semmelweis refuted it by arguing that the cause operated to the same extent in the two maternity clinics (Note 6), or by pointing out that their implications were absurd, as, for example, with the suggestion that rough internal examinations by foreign medical students was the cause of the disproportionately high MMR from CBF in Division I:

> If such a rough examination . . . may cause such a fearful condition as puerperal fever . . . passage of the fetal body through the genitals must be just as harmful, so that it cannot be imagined why each birth does not end fatally. (Semmelweis, p. 377)

None of the putative causes that Semmelweis investigated could explain the disproportionately high MMR from CBF in Division I, which left him dejectedly lamenting, "Everything was uncertain, everything was doubtful, everything was inexplicable, only the enormous number of deaths was an indubitable fact." (Semmelweis, p. 390). It was not until he had deduced the nature and cause of CBF that Semmelweis was finally able to formulate and test his hypothesis about the cause of the disproportionately high MMR from CBF in Division I.

2.1.2 Investigations during the Second Period of Semmelweis's Assistantship

2.1.2.1 Deductions about the Nature and Cause of Childbed Fever

Semmelweis learned of the pivotal event that would enable him to deduce the nature and cause of CBF on his return from a vacation in Venice that he took before resuming his position as First Assistant on March 20, 1847: the professor of forensic pathology, Jacob Kolletschka, had died after a student had accidently cut Kolletschka's finger during an autopsy. (Semmelweis, p. 390) Semmelweis examined Kolletschka's autopsy report and noted that the findings reported were, with the exception of the

internal genitalia, the same as those he had repeatedly observed at autopsies of women who had died of CBF. Relying on the first of his inductive generalizations or axioms, Semmelweis concluded that since the pathological changes in Kolletshcka and women who died of CBF were the same, the diseases that caused their deaths must also have been the same:

> From the *identity* of the *pathological findings* in the cadavers of the newborns with the pathological findings in the women who died of childbed fever, we had concluded earlier, and we think rightly, that the newborns also died of childbed fever, or in other words, the newborns died of the same disease as did the puerperae. Since we came upon the *identical* results in the *pathological findings* in Kolletschka as in the puerperae, the conclusions that Kolletschka died of the same disease, from which I had seen so many hundred puerperae die, likewise was justified. (Semmelweis, 1941, p. 392, italics added).

This was a logically valid *modus ponens* inference, the form of which is: If P then Q; P; therefore, Q. In Semmelweis's case, the major (conditional) premise (shown in italics) was suppressed, and the inference had following form: "the pathological findings are the same; *if the pathological findings at autopsy are the same, the diseases producing the changes must also have been the same*; therefore, the diseases are the same."

Having deduced that CBF was the same disease as the disease that had killed Kolletschka, Semmelweis relied on his second inductive generalization to conclude that the cause of CBF had to be the same as the cause of Kolletshcka's illness, which was known from the circumstances of Kolletschka's death:

> The exciting cause of Professor Kolletschka's illness was known, that is to say, the wound produced by the autopsy knife was contaminated at the same time by cadaveric material. Not the wound, but the contamination of the wound by cadaveric material was the cause of death. Kolletschka was not the first do die in this fashion. I must acknowledge, if Kolletschka's disease and the disease from which I saw so many puerperae die are identical, then in the puerperae it must be produced by the self-same engendering cause, which produced it in Kolletschka. In Kolletschka the specific agent was cadaveric particles, which were introduced into the vascular system. I must ask myself the question: Did the cadaveric particles make their way into the vascular systems of the individuals whom I had seen die of an identical disease? This question I answered in the affirmative. (Semmelweis, 1941, p. 392).

This is, again, a *modus ponens* inference with a suppressed major premise shown in italics: "the diseases are the same; *if the diseases are the same, their causes must be the same*; therefore, the cause of CBF is the same as the cause of the disease that killed Kolletschka."

Semmelweis accepted that cadaveric matter introduced into the blood stream could cause CBF because he wrote, "*the fact was known to me that decaying organic matter brought into contact with living organisms produced in them a putrefactive process"*. (Semmelweis, 1941, p. 393, italics added) The source of this information was most likely the experiments that Gaspard had reported in 1822 and 1824

showing that the injection of putrid matter into animals intravenously caused fever and multiple abscesses, i.e. pyemia. (Bullock, 1938, pp. 129-131). Semmelweis must have been aware of this work for he wrote in his book.

> Fergusson says that Gaspard and Cruvelhier have injected decomposed matter into the vascular system, and thereby these same inflammatory reactions were produced in animals as we find in puerperae. (Semmelweis, 1941, p. 690).

Indeed, Garspard's work seems to have been generally known in European academic obstetrical circles, because Karl Levy, Director of the Copenhagen Lying-in Hospital, wrote in response to being informed of Semmelweis's work, that "it is not to be doubted" that by the "the direct introduction of putrid matter into the organism . . . a condition can be produced, which has many resemblances to puerperal pyemia." (see below)

Semmelweis concluded that the nature of the disease from which Kolletschka had died was pyemia (i.e. septicemia with or without abscess formation) because the causative agent was introduced directly into Kolletschka's bloodstream by the cut to his finger, and, therefore, the locations at which pathological changes were observed at autopsy could not have been where the disease started, but were, rather, the consequences of the disease process. (Note 7) Since the disease producing the pathological changes in Kolletschka had to be the same as the disease that had produced identical pathological changes in mothers dying of childbed fever, Semmelweis concluded that CBF was also a form of pyemia. (Semmewelweis, p. 558: "Since the findings in cadavers dead from pyemia are identical with those dead of childbed fever, then childbed fever is the same disease.")

2.1.2.2 Cause of the Disproportionately High MMR from CBF in Division I

Once Semmelweis realized that decomposing matter from cadavers could cause CBF, he was able to formulate and test his hypothesis about the cause of the disproportionately high MMR from CBF in Division I through a chain of inferences that were based on two well-known facts: (1) that a characteristic cadaveric odor could linger for extended periods on the hands of those who had been engaged in autopsies even after they had washed their hands with soap and water, and that (2) medical students and student midwives were taught in different divisions, and only medical student were involved in autopsies. He could attribute no causal significance to these facts before he had deduced the nature and cause of CBF as CBF was believed to be a specific disease of the puerperium and there was no way to link it to autopsies.

From the characteristic cadaveric odor that lingered on the hands of those engaged in autopsies, Semmelweis hypothesized that those involved in autopsies could transport decomposed matter on their hands from the autopsy room to the labor ward and introduce it into the birth canals of women when they examined them in labor:

> That the cadaveric particles clinging to the hands are not entirely removed by the ordinary method of washing the hands with soap and water, is shown by the cadaveric odor, which the hands retain for a longer or shorter time. During the examination of gravidae,

> parturients, and puerperae, the hand contaminated with cadaveric particles is brought into contact with the genitals of these individuals, and hence the possibility of absorption, and by means of absorption, introduction of cadaveric particles into the vascular system of these individuals is *postulated*, and by this means the same disease is produced in these puerperae, which we saw in Kolletschka. (Semmelweis, 1941, p. 393).

Semmelweis concluded that once decomposed matter was introduced into the birth canal it could be absorbed into the blood stream through the raw surface of the placental bed—where the placenta had been attached to the uterus during pregnancy—and cause CBF:

> In gravidae, parturients or puerperae, there is a place in the body, which has no epidermis or epithelium, and that is the internal surface of the uterus; starting from the internal os upwards, this is the absorption place for the decomposed matter which causes puerperal fever. If wounds are caused by labour, then every place on the genitals, indeed any wound on the body, can be the site of absorption. (Semmelweis, 1941, p. 504).

The fact that medical students and midwives were taught on separate divisions and only the former were involved in autopsies could now easily explain why the MMR from CBF was so much higher in Division I: because decomposed matter from the autopsy room was introduced much more frequently into the birth canals of women in Division I than in Division II:

> *After* it became evident that the preponderance of mortality in the First Clinic in contrast with the Second was to be sought in the cadaveric and ichorous [purulent] particles, with which the hands of the examiners were contaminated, the previously inexplicable phenomena, which took place in the First Division, could be very easily explained. (Semmelweis, 1941, p. 401, italics added).

However, the cause of CBF, and the sources of the causative agent, were exactly the same in the two divisions, only the morgue was much less frequently the source of CBF in Division II. But it was the source of the causative agent in some cases as the assistants in Division II, even if not the student midwives, were involved in autopsies, and some, like Zipfel, performed autopsies quite assiduously. (Semmelweis, p. 688: "I saw Dr. Zipfel do autopsies very frequently on the puerperae who died at the midwives' clinic.") This was confirmed by Arneth in 1851 during his lecture before the Edinburgh Medico-Chirurgical Society. Arneth explained that there were much fewer cases of CBF in Division II after medical students and student midwives were taught in different divisions, "with one striking exception":

> the period between 15th October 1841 and 15th October 1843, when the mortality was much higher than usual, the then assistant [i.e. Zipfel] being very busy in anatomical pursuits (i.e. performed many autopsies). (Arneth, p. 509).

2.1.2.3 Proof of Semmelweis's Causal Hypothesis

The abductive inference about the cause of the higher mortality rate in Division I was a hypothesis that had to be proved, which Semmelweis sought to prove by destroying the cause chemically to determine if this would prevent the disease:

> If the hypothesis is correct . . . this disease can be prevented *to the extent* that it is dependent upon the effect of cadaveric particles carried by the examining finger . . . In order to destroy the cadaveric particles adhering to the hand . . .I began to use "Chlorina liquida" . . .[but] after some time . . . changed to the considerably cheaper chlorinated lime. (Semmelweis, 1941, p. 387, italics added)

The is a *modus tollens* inference, the logical form of which is, If P, then Q; not-Q; therefore, not-P, and Semmelweis was quite explicit that the CH-D experiment was designed to prove his hypothesis about the *cause of the disproportionately high MMR on the first maternity clinic*—i.e. his causal hypothesis—not the cause of CBF in general, which was a deductive inference that Semmelweis accepted as true:

> I have assumed that the cadaveric particles adhering to the examining hand of the accoucheur is the *cause of the greater mortality in the First Obstetrical Clinic*; I have eliminated this factor by the introduction of the chlorine washings. The result was that the mortality in the First Clinic was confined within the limits of that in the Second, as the above cited figures show. The conclusion, therefore, that the cadaveric particles adhering to the hand in reality *caused the preponderance of mortality in the First Clinic*, was *also* a correct one. (Semmelweis, 1941, p. 395, italics added).

3. Tulodziecki's Erroneous Representations about Semmelweis's Work

Tuodziecki's revisionist account of Semmelweis theory and reasoning contains the following seven major misconceptions.

Misconception 1: Semmelweis propounded three versions of his theory

Tulodziecki's contended that Semmelweis propounded three versions of his theory, which were simply an embellished versions of Irwine Loudon's ersatz account of Semmelweis's discovery, (Note 8) and which essentially confuses the causative agent itself with the source of the causative agent:

> it is important to understand that he [Semmelweis] went through three successive versions of his theory: first a version according to which cadaveric matter from corpses was responsible for childbed fever [version 1—the so called 'cadaveric theory']; second a version according to which, in addition to cadaveric matter from corpses, people somehow produced their own cadaveric matter, which could also cause the disease [version 2]; third, a version according to which any kind of decomposing animal matter could cause the disease [version 3]…

> After Semmelweis realized that version 1 could not be quite right he proceeded to amend his view to claim that in those cases in which no link to a corpse could be established, the nondiseased body must somehow produce its own cadaveric matter, and in this way cause the disease [version 2]. Semmelweis was not very specific about how this might happen; yet, once again, he insisted quite dogmatically that *these two routes exhausted the possible causes*. Upon realizing that there were problems with this view as well, he finally arrived at his final version according to which any decomposing animal matter could cause the disease, as long as it was introduced into the genital tract [version 3].' (Tulodziecki, 2013, p. 1072, italics added).

This account of Semmelweis's theory has no basis in historical facts. Semmelweis never at any time defined all cases of CBF as caused by 'cadaveric particles' (Tulodziecki's 'version 1', or Loudon's 'cadaveric theory'), and Tulodziecki cited no evidence or statements by Semmelweis to the contrary (nor did Loudon). The phrase 'cadaveric particles', like the phrase 'cadaveric matter, which Semmelweis also used, merely referred to the source of the causative agent that was introduced into Kolletschka's blood stream when a student accidentally cut Kolletschka's finger during an autopsy. As previously noted, Semmelweis accepted that cadaveric matter introduced into the blood stream could cause CBF because he was aware that pathological changes resembling "puerperal pyemia" could be induced in animals by introducing decaying organic matter into their blood stream.

No historical evidence supports Tulodziecki's contention that Semmelweis "realized that Version 1 [of his theory] was not quite right". On the contrary, Semmelweis repeatedly reaffirmed throughout his book that there were three sources of decaying animal-organic matter that caused CBF, cadavers being only one of them. (Note 9) Nor did Semmelweis's realization that the causative agent could come from living persons as well as cadavers have anything to do with the correctness or incorrectness of 'Version 1' but was inferred from the 'row infections' that followed the admission of a patient with an infected carcinoma of the uterus in October, 1847.

Because cadavers were the only known source of decaying animal organic matter when Semmelweis implemented the chlorine hand-disinfection (CH-D) experiment at the end of May, 1847, Semmelweis only required attendants to disinfect their hands before entering the labor ward, but once on the ward they were permitted to wash their hands with soap and water as before. However, in October, 1847 a pregnant woman with an infected carcinoma of the uterus was admitted to the labor ward, and placed in the bed by the door at which ward rounds always started. After examining this patient, students examined each of the twelve patients who were in the row of beds along the ward, and eleven of them developed CBF. These 'row infections' made Semmelweis realize that the causative agent could come from living persons with 'ichorous' (purulent) discharges as well as cadavers, and he immediately required attendants from then on to disinfect their hands with chlorine solution before examining *any* patient in labor. (Semmelweis, 1941, p. 396).

Nevertheless, another outbreak of CBF occurred the following month after a pregnant woman with a knee infection was admitted to the ward, and Semmelweis concluded from the odor in the room, and the fact that mothers in the same room as this patient developed CBF, that decaying animal-organic matter could be carried by air, and that patients with open infections could cause the air in a ward to become so saturated with decaying matter that they had to be isolated. However, the air was simply the vector and not the cause, which was still 'decomposed matter' in these rare cases. (Semmelweis, 1941, pp. 396-297)

Semmelweis subsequently concluded that decomposing animal-organic matter that reached the mother's blood stream and caused CBF could originate from the mother's own reproductive tract (Tulodziecki's 'Version 2') (Note 10). But, again, Semmelweis also did not formulate his theory of autoinfection after he "realized that Version 1 could not be quite right" (Tulodziecki, p. 1072) but to explain why there was an irreducible minimum number of cases of CBF that CH-D could not eliminate, and which he estimated should be at most 1%, based on the MMR from CBF at the AKH prior to the introduction of routine autopsies in1823. (Semmelweis, 1941, pp. 435-437). And far from being vague about it, Semmelweis was very specific about how decaying organic matter could be generated within the mother's own birth canal, and cause CBF:

> As for the etiological factors which cause the formation of a decomposed matter within the individual and thereby causes childbed fever through auto-infection, they are the following: Decomposition of the normal lochial flow, as a result of a rather prolonged retention of the lochial discharge from whatever cause, retention of blood clots in the uterine cavity after haemorrhage, retention of the placenta or placental and membranous remnants, bruising of the genital organs as a result of a prolonged stage of expulsion, or as a result of necrosing perineal lacerations after operations. (Ibid., p. 552).

In other words, Tulodziecki confused cause and source, and equated the three different sources of the same causative agent that Semmelweis's identified as three different causes, which they were not. They were three sources of decaying animal organic matter.

Misconception No. 2: Semmelweis's final theory ('Version 3') was not taken seriously because he had already twice before insisted that he had found the only cause of childbed fever.

Tulodziecki contended that 'Version 3' of Semmelweis theory—what she construed as his final version—was not taken seriously because Semmelweis "had already, unsuccessfully, insisted *twice before* (sic) that he had identified the only cause of puerperal fever". Again, Tulodziecki cited no authority for these claims, which are contradicted by the publications about Semmelweis's work that prove that nothing was communicated about Semmelweis's theory before 'Version 3' was published in December, 1847, and, therefore, Semmelweis could not have insisted twice before that he had identified the only cause of CBF.

The results of Semmelweis's CH-D experiment were first published in an Editorial by Hebra in December, 1847, (Semmelweis, 1941, pp. 561-562), and privately on December 21, 1847 by Hermann

Schwartz in a letter to his chief in Kiel, Gustav Michaelis, (Carter & Tate, 1991, p. 256). There were no earlier private or public communications about Semmelweis's work, and far from not taking this 'version' seriously, Michaelis informed Semmelweis in his reply to Schwartz's letter that he had eliminated CBF in his hospital by implementing CH-D, and mentioned a source of the causative agent other than cadavers, namely an improperly cleaned catheter. (Semmelweis, 1941, p. 569) The next publication about Semmelweis's work was in November 1848 in England by Routh, who informed Semmelweis that his lecture was well received by an audience that included some of the most prominent obstetricians in England. (Ibid., p. 566) Although Routh did not mention the events that led Semmelweis to conclude that decaying animal organic matter could come from living persons with purulent discharges, he did refer to CBF being caused by "the direct inoculation of poisonous secretions derived from gangrenous wounds." (Routh, 1848, p. 36)

Friedrich Wieger from Strasbourg next reported on 'Version 3' of Semmelweis's theory in April, 1849, and he also mentioned a non-cadaveric source of the cause of CBF by describing a case of CBF caused by a midwife's use of a poorly washed sponge to clean the mother's perineum that led to the revocation of the midwife's licence. (Kadar & Croft, 2020, p. 391). Although the next publication about Semmelweis's work, Skoda's lecture to the Imperial Academy of Science on October 18, 1849, only mentioned cadavers as the source of the causative agent, Skoda made it unambiguously clear that by 'cause'—which Skoda referred to as rotting or putrid animal matter and not as 'cadaveric particles'—he was referring to the cause of the disproportionately high MMR from CBF on Division I and not to the cause of CBF *tout court*, and that putrid matter was only one of the possible causes of CBF that was simply less operative on Division II:

> This was at the same time *only one among the possible causes of puerperal disease*, which on the division for midwives was either not at all effective or extremely limited, so that, assuming this cause, the very unequal number of diseases in the two departments would be very understandable. (von György, 1905, p. 39, italics added).

Semmelweis himself first reported on his theory in the following year, in a lecture he delivered before the Viennese Society of Physicians on May 15, 1850, and in that lecture Semmelweis made it clear that the rotting or putrid matter Skoda had referred to could come from sources other than cadavers, for the minutes of the meeting state in relevant part:

> the continued new introduction of such substances, especially on the first maternity clinic, by the assistants and students must be accepted based on the daily dissection of corpses, *but without excluding other modes of transfer of rotting organic constituents to the maternal organism such as decomposed placental remnants, continuous touching of the sick and healthy pregnant and newly delivered women, and dissolution of fluids in other patients.* (von György 190, 49, italics added).

This was reaffirmed the next year by Arneth in his lecture before the Edinburgh Medico-Chirurgical Society, in which Arneth (1851, 508, italics in original) stated:

> The opinion of Dr. Semmelweiss (sic) on this important matter is as follows: *Any fluid matter in a state of putrefaction . . . may produce puerperal fever*.

Arneth's lecture was well received and Simpson found it to be persuasive (see below). So no one, much less Semmelweis, published any other theory besides the theory that Tulodziecki labelled Version 3 prior to 1850, when Semmelweis reiterated that the causative agent of CBF could come from sources other than cadavers.

Misconception No. 3: Semmelweis claimed repeatedly that cadaveric matter from corpses was the only possible way for women to contract childbed fever.

Despite the fact that Semmelweis never at any time even suggested, much less claimed, that "cadaveric matter from corpses was the only possible way for women to contract childbed fever," Tulodziecki contended that Semmelweis repeatedly (sic) claimed that "cadaveric matter from corpses" was the only way women could contract CBF:

> According to version 1, cadaveric matter from corpses was the only possible way for women to contract childbed fever. It is worth noting that, even with respect to this version, Semmelweis was quite dogmatic and insisted on monocausality, *claiming repeatedly* that cadaveric matter from corpses was the only possible way for women to contract childbed fever. It is quite easy to see why Semmelweis's contemporaries did not accept this. (Tulodziecki, 2013, p. 1071, italcs added)

Tulodziecki cited no evidentiary basis for these statements that imputed to Semmelweis a view he never espoused. In fact, Semmelweis detailed clearly the three different *sources* of decaying animal organic matter in his book, which were:

> (1) "cadavers of every age, of either sex whose disease brings about the production of decaying animal organic matter;" (2) "sick persons of every age, of either sex, whose illness brings about the production of decaying animal organic matter;" and (3) "physiologic animal-organic structures (i.e. bodily fluids and discharges) which, no longer subject to the vital laws, attain a certain degree of putrefaction; not what the structure is but the degree of putrefaction is to be considered"; and, rarely (in at most 1% of cases), the source of the decaying animal organic matter could also be the mother's own genital tract, which Semmelweis referred to as "autoinfection". (Semmelweis, 1941, pp. 429-430)

And as previously noted, Semmelweis repeated throughout his book that there were three sources of decaying matter, cadavers being only one of them. (*See supra* note 7).

However, Semmelweis's clearest and most emphatic statement that he had never espoused such a view is contained in his open letter to Eduard Caspar Jacob von Siebold, a distinguished professor of Obstetrics at Göttingen, who had visited Semmelweis in Vienna and in Pest, and had read Semmelweis's book. Nevertheless, in an article published in 1861, von Siebold imputed to Semmelweis what Tulodziecki called a 'cadaveric theory', and the following was Semmelweis's reply:

> And if you, Herr Hofrath, in spite of all this attribute to me a doctrine which ascribes all cases of puerperal fever to infection from the cadaver, you are either intentionally misrepresenting my doctrine or your understanding of it is flawed. (von Györy, 1905, p. 445) (Note 11)

Misconception No. 4: Semmelweis could not explain the seasonality of CBF

Tulodziecki contended, as had Loudon, that one of the reasons that Semmelweis's theory was rejected was that he could not explain the seasonality of CBF:

> there were also some more general reasons why people were suspicious of Semmelweis's claim that decomposing (or even just cadaveric) organic matter was the only cause of childbed fever. For example, it had been known for a long time that childbed fever was seasonal, with its highest mortality rate in late winter, usually February, and a low in August. Semmelweis simply denies that this is the case. He does give what he thinks is an explanation of the apparently seasonal patterns . . . However, once again, this was not viewed as a convincing argument by Semmelweis's contemporaries since, in fact, childbed fever was highly seasonal . . . (Tulodziecki, 2013, pp. 1073-1074)

Semmelweis adduced the following evidence to support his contention that the cause of CBF was not seasonal, which Tulodziecki, like Loudon, either ignored or was unaware of:

> 1) Semmelweis (1941, p. 442) noted that "[i]t is the prevailing opinion that winter is the season which principally favors the outbreaks of childbed fever . . . [b]ut this phenomenon is not to be explained by the atmospheric influences of winter for otherwise childbed fever could never occur to a greater extent in summer," but Semmelweis demonstrated that every month of the year had, in different years, the highest or the lowest MMR from CBF for that year at the AKH. For example, the MMR from CBF for the year 1844 was lowest in June, where as it was the highest in June in the year 1846. (Semmelweis, 1941, p. 360, Table II) Therefore, the MMR was not always highest during winter months;
>
> 2) During the 39 years before autopsies became routine at the AKH (1823), the MMR was less than 1% in 25 of those 39 years, which would have to mean that if CBF only occurred during the winter, there were no winters in Vienna for 25 years (ibid., p. 444);
>
> 3) Every year, epidemics of CBF ravaged St. Rochus Hospital in Pest, but obstetrical patients were only admitted to that hospital in August and September of each year, when the university was on vacation (ibid.)

Semmelweis also explained that the tendency for the MMR from CBF to be higher during the winter months at the AKH was not due to the seasons *per se*, but attributable to two factors. Tulodziecki, like

Loudon, mentioned only the less important of the two factors, namely, that students attended to their studies more assiduously during the winter months than during the summer. (Tulodziecki, 2013, p. 1073). However, a more important factor was that during the winter autopsies were always performed *before* the 4PM ward rounds on the maternity clinic because it was too dark later in the day to perform autopsies, whereas in the summer months, when it was still light after the 4PM ward rounds ended, autopsies tended to be performed *after* the ward rounds, as it was too hot, and the smell in the autopsy room too oppressive to perform autopsies earlier in the day (Semmelweis, 1941, p. 444) In other words, women in labour were more likely to be examined after an autopsy in winter than in the summer months.

Misconception No. 5: Semmelweis denied that childbed fever occurred in epidemics.

> He [Semmelweis] also insisted that puerperal fever was not epidemic, which, again, was simply false. (Tulodziecki, 2013, p. 1074)

Tulodziecki conflated the two senses in which the word "epidemic" was applied to CBF in the above statement. One sense, and the usual one, refers to the number of individuals affected by the disease in question. Semmelweis never denied that the frequency of CBF in a locality could abruptly increase and rise to 'epidemic' proportions in the first sense of the word 'epidemic'. He made it crystal clear that what he meant by stating that the cause of CBF was not epidemic was that it was not caused by some characteristic of the air, i.e. the second sense in which the word epidemic was applied to CBF to refer to its cause. Semmelweis considered the number of cases involved irrelevant to their cause:

> In advancing the concept of a puerperal epidemic and endemic, one must disregard in its entirety the number of ill and deceased puerperae . . . An epidemic puerperal fever is one which is brought about through atmospheric-cosmic-telluric influences and whether one or a hundred patients die is immaterial in the concept of an epidemic. (Semmelweis, 1941, p. 389)
>
> The concept of the epidemics is given by the cause . . .independently of the number . . . (Ibid., p. 441).

Misconception No. 6 about Semmelweis's animal experiments.

Tulodziecki's sixth misconception embodies a number of misconceptions about Semmelweis's animal experiments for Tulodziecki misconstrued (1) how many sets of animal experiments Semmelweis conducted; (2) why he conducted animal experiments; (3) who asked Semmelweis to conduct a second set of experiments and why; and (4) why Semmelweis did not conduct a third set of experiments.

> There was, however, another piece of evidence Semmelweis introduced. This was related to his claim that all childbed fever is really a species of pyemia . . . In support of this view, Semmelweis provided two main arguments. The first was an argument relying on the only series of animal experiments he ever performed . . . Moreover, he claimed that the experiments proved this with certainty. The problem with this argument, however, was that . . . *people* objected that what Semmelweis had shown, at most, was that introducing

> pyemic matter into rabbits could produce pyemia in rabbits, but that this was not what needed to be shown . . . In fact, *people were quite specific* about outlining the kinds of experiments they would have liked Semmelweis to perform, all of them essentially—and quite reasonably involving injecting the rabbits with matter from actual victims of childbed fever, not other diseases. Semmelweis's only response, however, was to claim that, in his opinion, such experiments were superfluous. (Tulodziecki, 2013, p. 1073)

First, Semmelweis conducted two sets of experiments not one. (Kadar, 2020) He conducted the first set of experiments with Rokitansky's assistant, Georg Maria Lautner to demonstrate that "the injection of deleterious material into the vagina is capable of causing pyemia." (Semmelweis, 1941, p. 613). Skoda described each experiment during his October 18, 1849 lecture to the Academy of Science on Semmelweis's work, and acknowledged that the experiments had achieved their purpose:

> It is barely necessary to mention that the changes encountered in the corpses of the rabbits were the same as that resulting from puerperal fever, and, in general, from pyemia in human corpses. (von György, 1905, p. 45)

Semmelweis conducted a second set of experiments in the Spring and Summer of 1850 at the behest of Skoda, who told the Academy of Sciences that doubts remained about Semmelweis's theory, and asked the Academy to award Semmelweis a grant to conduct 'further and more varied animal experiments' with the physiologist Ernst Ritter von Brücke, a member of the Academy, to remove any doubt that they were conducted properly:

> Therefore, I ask that you be willing to support Dr. Semmelweis in further experiments with a grant of money, and in view of this, in order to dispel any doubts as to the correctness of the experiments, that these attempts are also made by a member of the Academy, I ask Professor Brücke to undertake this task. (von György, p. 45)

However, Skoda never identified what the remaining doubts about Semmelweis's theory ostensibly were, why the experiments Semmelweis had already conducted with Lautner had failed to resolve those doubts or what kind of more varied experiments would allay the remaining doubts or why. (Kadar, pp. 394-395) Moreover, Brücke informed the Academy in a memorandum that he had concluded from the ambiguous results obtained with this second set of experiments that animal experiments were not the way to remove any remaining doubts about Semmelweis's theory, and that this could be only done by collecting additional clinical evidence:

> Last autumn, at the request of Professor Skoda, this esteemed Section (Klasse) asked me to conduct experiments in animals with Dr. Ignaz Semmelweis, in view of his statements about how puerperal fever came into being… Dr. Semmelweis had already in the spring and summer made these attempts with great zeal and conscientiousness, and carried out the post-mortems on the animals with me. These have, however, until now delivered only ambiguous result, and have convinced me that experiments on animals are not the suitable means to lift the doubts about this important . . . topic . . . [and] that this can only happen

> through the collection of experiences similar to those Dr. Semmelweis made in the local obstetrical institution . . . (von György, 1905, pp. 288-289)

By the time he wrote his book, Semmelweis had reduced the MMR from CBF at two different hospitals in Pest—to 0.85% at the Rokus Hospital, and to 0.39% at the University of Pest Hospital—which caused Semmelweis to comment after he had quoted Brücke's memorandum

> Since then [i.e. the experiments he had conducted with Brücke] I have had similar experiences as in Vienna in two other institutions; I have also compiled in this text the similar experiences of others, and believe that as a result of the full impact of these experiences, experiments on animals are superfluous. (von Györym, 1905, p. 289)

Tulodziecki appears to have conflated the history of Semmelweis's animal experiments with the response that Karl Levy gave when he was first notified of the results of Semmelweis's CH-D experiment in 1848, and seems to have been unaware that it was Skoda, not Levy, who asked Semmelweis to conduct further animal experiments. As previously noted, Michaelis's assistant had described Semmelweis's CH-D experiments in a letter to Michaelis dated December 21, 1847, and Michaelis forwarded this letter to Levy, who then published his response to the letter, together with Schwartz's letter, in a Danish medical journal of which Levy was an associate editor. In his reply to Hebra's Editorial and Schwartz's letter, Michaelis indicated that he had sent a copy of Schwartz's letter to Levy, which prompted Semmelweis to write to Levy and ask him to comment on Semmelweis's findings. However, Levy did not reply to Semmelweis until ten years later (May, 1858) as he had assumed that Michaelis would forward a translation of his comments on Semmelweis's work to Semmelweis, but this did not happen because Michaelis committed suicide. (Semmelweis, 1941, p. 572)

Levy's response is noteworthy for two reasons, but he never asked Semmelweis either expressly or impliedly to conduct animal experiments using only material from "puerperal cadavers", i.e., women who had died of CBF. He merely stated that "a strict examination would absolutely require that this *difference in the source* of the infection [i.e. disease that the cadavers had died of] . . . be taken into consideration." (Semmelweis, p. 574, italics added). However, Levy went on to write:

> In the cases where the infectious matter may originate in any sort of a cadaver, every idea of a specific contagium must be abandoned, and in place of this, the infection of the blood-mass, in so far as such a thing occurs, must be classed along with the blood infection brought about by the direct introduction of putrid matter into the organism by many experiments on animals. *That hereby a condition can be produced, which has many resemblances to puerperal pyemia, is not to be doubted* . . . (Semmelweis, 1941, p. 575, italics added)

Exactly so, and this was precisely the inference that Semmelweis drew: that CBF was a form of pyemia and not a specific disease of the puerperium as Rokitansky believed, that could be caused by the introduction of animal-organic putrefying matter from *any* source into the blood stream, and not by a

specific contagium as the British maintained. Furthermore, Semmelweis accepted that the introduction of putrefying matter into the blood stream could cause pyemia on the basis of the very experiments Levy alluded to, and that originated with Gaspard in France, as previously noted.

It seems that Tulodziecki misconstrued this reference to animal experiments that had already been conducted decades previously in France by Gaspard as a request by Levy for Semmelweis to conduct further animal experiments. (Note 12) However, Levy never expressly made such a request, nor could his reference to animal experiments be construed as an implied request that Semmelweis conduct further animal experiments for Levy acknowledged that it could not be doubted that one could produce a condition that resembles 'puerperal pyemia' by introducing putrid matter into the organism. Levy suggested that making clear *the source of the putrid matter* was important because it might explain why "puerperal fever manifests itself under many other forms." (Semmelweis, p. 585, italics added). Semmelweis, was well aware of this fact but suggested different reasons for it:

> Why the decomposed, absorbed matter causes pyemia at one time, and the other forms at others we do not know. Perhaps the reason lies in the different degrees of putrefaction of the decomposed matter, perhaps in the different reactive susceptibilities of the organisms. (Semmelweis, pp. 575-576).

Myth 7: Semmelweis's views about how childbed fever was spread were not new and preceded by eleven years by the views of the British contagionists.

Although Tulodziecki maintained that there was no paradigm about the cause of CBF, her main argument was that even if there was a paradigm, Semmelweis did not contradict it because Semmelweis's views were the same as those of the British contagionists, only their theories, and specifically the theory of the Scottish obstetrician, James Young Simpson, preceded Semmelweis's theory of causation by 11 years:

> Semmelweis realized that it [the cause of CBF] could be introduced into the genital tract through the hands of medical staff, whereas others thought it was conveyed through air. This view, however, was not new, either. James Young Simpson, a Scottish obstetrician, for example, was specific about how childbed fever could be contracted, publishing the following [which stated in relevant part that "patients during labour . . . may be locally inoculated with a materies morbi capable of exciting puerperal fever] 11 years before Semmelweis. (Tulodziecki, 2013, p. 1069)

These statements are also factually incorrect for Simpson's views did not precede Semmelweis's theory, much less precede it by 11 years. Semmelweis's results were communicated personally to Simpson in 1848 by Franz von Arneth, (Semmelweis, pp. 565-566); they had also been reported in two articles prior to 1850 by Routh and Wieger (see above), and Simpson was aware of these articles for he cited them in a footnote to his 1850 article. (Simpson, 1950, n. 1, p. 429.) Simpson also mentioned Semmelweis by name in that article, and acknowledged his discovery:

> By 1847 almost every woman delivered in the wards [of Division I] attended by the medical students, was examined by a number of students; and these students had been often allowed immediately previously to touch and handle the bodies of women who had died of puerperal fever, and were even taught upon them the manipulations and operations of midwifery. The mortality altered and diminished immensely and immediately from the time (May, 1847) that the assistant-physician, Dr. Semmelweiss (sic), prevented students from touching parts of the autopsies, and directed all of them to wash their hands in a solution of chorine before and after every vaginal examination. (ibid., p. 429).

Simpson also later acknowledged and accepted Semmelweis's theory as the following comments on Arneth's presentation of Semmelweis's work indicates:

> Dr. Arneth's very valuable paper adduced what was apparently incontrovertible evidence of puerperal fever being propagated in the way he suggested . . . (Simpson, 1850, p. 506).

It is certainly true that Gordon identified that CBF could be conveyed to healthy women in labor by their attendants before Semmelweis, and that subsequently several British obstetricians made the same observation before Semmelweis made his discoveries (Parsons, p. 148), but what they, unlike Semmelweis, were unable to do was to work out the mechanism by which the disease was caused and to formulate an explanatory hypothesis for their empirical observations. On the contrary, although Gordon maintained that puerperal fever is "a special contagion or infection altogether unconnected with the noxious state of the atmosphere," he nevertheless held that it was communicated by an atmosphere charged with infections, and never correctly identified the vectors of the disease:

> I had evident proofs that every person, who had been with a patient in the puerperal fever, became charged with an atmosphere of infection, which was communicated to every pregnant woman, who happened to come within its sphere. This is not an assertion but a fact, admitting of demonstration, as may be seen by a perusal of the forgoing table. (Gordon, 1795, pp. 63-64)

Gordon's assertion was an erroneous inference, not a 'fact'. Moreover, Simpson only realized that wound infections and CBF were analogous diseases after he was apprised of Semmelweis's work: he did not come to that conclusion based on Gordon's observations, and his immediate response when apprised of Semmelweis's work in 1848, was, according to Semmelweis, "filled with abuse". (Semmelweis, 1941, p. 565) As the distinguished German gynaecologist and Semmelweis's first biographer, Alfred Hegar, put it:

> Simpson . . . misunderstood Semmelweis's doctrine completely, which he considered to be identical to the English doctrine of a specific contagion, and claimed that Semmelweis did not discover anything new and that everything had long been known in England. Nevertheless, apparently influenced by Semmelweis's reports, the same scholar later published two essays in which the etiology of puerperal fever was understood almost entirely as Semmelweis described it, and the disease was also lumped together with

> pyaemia (surgical fever). Simpson has been most unjustly credited as being the first to describe childbed fever as a surgical fever and newly delivered women as 'wounded'. This was first done by Cruveilhier, and more incisively (*prägnant*) by Semmelweis. (Hegar, 1882, pp. 18-19). (Note 13)

4. Discussion

Tulodziecki's statement that it is "important to prevent the mythical version of Semmelweis's story from being invoked as support for further philosophical theses," (Tulodziecki,) is ironic for two reasons. First, the accounts of Semmelweis's work in the philosophy of science literature are riddled with misconceptions, but they are not the ones Tulodziecki was referring to or, indeed, seems to be aware of. Second, Tulodziecki's account of the "Semmelweis story" has only added to the misconceptions as her historically untenable account of Semmelweis's theory and reasoning in incorrect in every material respect.

Why Semmelweis's theory about the nature and mechanism of causation of CBF was rejected by many of his contemporaries, given that they were correct, and how Semmelweis responded to his critics, are assuredly important historical questions to which answers are long overdue. However, to answer these questions accurately requires a careful and detailed analysis of all the available primary evidentiary sources, which philosophers of science are not well suited to undertake. The reason is that philosophers are not really interested in history for its own sake, and have merely used what happened in the past to support their present theories. Louis Mink (1965, p. 25) drew attention to this problem in the philosophy of history more than 50 years ago, and Scholl & Räz (2016, p. 11) belatedly admitted that this is also true of the interest that philosophers have had in Semmelweis's work, when they wrote: "For the most part [philosophers of science] are not primarily interested in Semmelweis *qua* Semmelweis: the topic of interest is confirmation, of which Semmelweis is taken to be a representative instance." Tulodziecki was also not interested in Semmelweis's work for its own sake; her purpose in reconstructing Semmelweis's work was, as previously noted, to refute Gillies's suggestion about why Semmelweis's theory was rejected by his contemporaries.

Historians have long recognized the dangers inherent in reconstructing the past for the sake of some present purpose. Butterfield (1965, pp. 30-31) denounced it as "the source of all sins and sophistries in history;" the philosopher and historian, Michael Oakeshott, saw the problem as being that when the past is reconstructed instrumentally, it is read backwards from the present, and only those past events are recognized that can be related to what is presently of interest: (Note 14) i.e. the instrumental use of history is fraught with confirmation bias, which influences how evidence is selected for consideration from all the available evidence. Under the sway of confirmation bias, evidence is selected to fit pre-existing theories rather than have the theories fit the historical evidence, and evidence inconsistent with the theory under consideration is discounted or ignored entirely. Tulodziecki's revisionist account of Semmelweis's work represents the apogee of these dangers for her account of Semmelweis's theory

and reasoning is not only incorrect in every material respect, it also fails to capture the diversity of views held about Semmelweis's work among his contemporaries or the extent to which his theory was actually rejected.

First, Semmelweis's theory was by no means universally rejected during his lifetime. Influential members of the Vienna Medical School—Hebra, Skoda, Rokitansky, Haller, Dumreicher—supported Semmelweis (Semmelweis, 1941, pp. 561-564, p. 702; Lesky (1964), pp. 39-40, pp. 87-89), as did other professors both in Vienna—Helm, Chiari, Arneth (Hegar, 1882, p. 19) —and outside Vienna—Michaelis, Lange, Kugelman, Oppolzer, Hirsch, Veit, Winckel, Liebig (Hegar, 1882, pp. 33-34; Semmelweis, 1941, p. 676) —and Semmelweis's work was endorsed enthusiastically in St. Petersburg. (von Györy, 1905, pp. 512-538). Furthermore, some opponents, like Späth, who was appointed professor in Division II of the AKH in 1861, did a *volte face* during Semmelweis's own lifetime, accepted the correctness of Semmelweis doctrine based on his own data showing that the MMR from CBF was much higher among hospital deliveries than among 'street births', (Lesky, 1976, p. 190), and wrote in 1864, "I also venture to state unreservedly that there is no longer any obstetrician, who is not most deeply convinced of the correctness of Semmelweis' views . . ." (Murphy, 1946, pp. 669-670). Even Scanzoni, one of Semmelweis's bitterest opponents, acknowledged the correctness of Semmelweis's doctrine in the fourth edition of his textbook, published shortly after Semmelweis's death in 1867, in which Scanzoni wrote:

> Puerperal fever is an infection caused by the admission of products of putrid decomposition of animal matter into the blood . . . Furthermore, we cannot and will not leave unmentioned the fact that, by his restless and self-sacrificing efforts in this field, Semmelweis has rendered a great service to lying-in women in our hospitals. It is fundamentally due to his efforts that in these institutions more attention than heretofore is being paid to the requirements of modern hygiene . . . (Gortvay & Zotán, 1968, p. 173)

Second, those who rejected Semmelweis's theory did not reject it for the same overarching reason as Tulodziecki's account suggests. For example, Semmelweis's chief, Johann Klein, rejected Semmelweis's conclusion about the cause of CBF because he did not accept that the reduction of deaths from CBF in 1848 was the result of the CH-D experiment, but believed that the MMR from CBF fell because the epidemic he believed was the cause of CBF had abated:

> It is by no means clear from the fact that since the termination of the last epidemic of childbed fever that the lower mortality rate is the result of chlorine washes, that the cause of the childbed epidemic has been found, and its recurrence was prevented. For this the experiment is still too young . . .(Note 15)

Eduard Lumpe, who was First Assistant in Division I during the period 1840-1842, also did not accept Semmelweis's conclusion that all cases of CBF were caused by the absorption of decaying animal-organic matter into the blood stream, because he maintained that the wide fluctuations in the incidence of CBF precluded that CBF had only one cause:

> During my period of service, there was such a difference between the maximum and minimum of mortality, that on could think of nearly everything else but a common invariable cause. (Semmelweis, p. 694).

However, Lumpe accepted that CH-D might be beneficial, and, therefore, recommended that until the cause of CBF is elucidated "we shall wait and wash." (Semmelweis, p. 701)

Third, the debates about Semmelweis's theory played out "within a context of animated professional rivalries . . . [and] ruinous competition for prestige and patronage," (Parsons, p. 138, p. 146). Semmelweis's most bitter and vociferous opponents rejected his theory of the etiology of CBF because it conflicted with their own theories and not because they genuinely misconstrued his theory as imputing all cases of CBF to 'cadeveric infection'. (Hegar, 1882, p. 20, p. 30) For example, Virchow, who also rejected the germ theory, rejected Semmelweis's theory of the cause of CBF because he maintained that thrombosis, caused by contraction of the uterus and surrounding blood vessels, was the cause of CBF, which could arise without external infection or injury when cold predisposed the body to general weakness. (Ibid., p. 31) Although it is certainly true that some did rejected Semmelweis's theory in the genuinely mistaken belief that he attributed all cases of CBF to cadaveric infection, (see e.g. Schürer von Waldheim, 1905, p. 55) this was not on account of anything Semmelweis ever wrote, but largely due to the statements Skoda made about Semmelweis's theory in his lecture to the Academy of Science, as previously argued by this author. (Note 16) Hegar was also likely correct that many professors of obstetrics were reluctant to accept Semmelweis's theory because it implicated them in causing the deaths of hundreds of young mothers, and they feared this would diminish their own prestige:

> his colleagues, by accepting his teaching, had to admit a certain amount of guilt. They had to say to themselves: You have often caused, even if unknowingly and only by following the opinions of your time, serious illness and death in your fellow men and, at least as concerns academic professors and authors, by spreading false doctrines, you have caused a much higher degree of suffering from such misfortunes. (Hegar, 1882, p. 46) (Note 17)

A comprehensive account of all the reasons that Semmelweis's theory was rejected by many, but by no means all of his contemporaries during Semmelweis's lifetime, is sorely needed but space limitation places this undertaking outside the scope of this article. However, the general points canvassed above should be sufficient to demonstrate that his theory and reasoning was rejected for a number of different reasons, and not one overarching reason, and Semmelweis's faulty reasoning was certainly not one of these reasons.

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Notes

Note 1. Childber fever is the literal translation of the term for puerperal fever in German (*Kindbettfieber*) and Hungarian (*Gyermekágyi láz*).

Note 2. Lesky (1976), 141.

Note 3. Ibid., 185: "[t]he fact that in 1844 Rokitansky made available to him all female cadavers at the pathological institute was undoubtedly of great importance to Semmelweis." Semmelweis also examined and dissected female cadavers who had died of gynecological diseases to teach himself gynecology, as gynecology was not part of the maternity divisions of the AKH, and was not taught to medical students, and had gained considerable expertise as an anatomic (gross) pathologist by the time he was appointed First Assistant. Lesky (1964), 91-92.

Note 4. This empirical generalization was likely the corollary of a principle Semmelweis learned from Rokitansky, who maintained that the anatomical changes observed at autopsies of individuals dying from various diseases were not givens but produced by disease *processes*, and wrote in his Handbook of Pathology: "anatomic alternations of organs and tissues are obviously the result of processes [and] an exact knowledge of these results [i.e. the pathologic alterations] was indispensable for acquiring an *insight into the nature of these processes*". (Klemperer 1961, 377, italics added).

Note 5. This assumption was a radical departure from the majoritarian view of Semmelweis's day when diseases were thought to have multiple causes. Carl Braun, for example, who succeeded Klein as professor on the first maternity division of the AKH, listed thirty causes of CBF. (Semmelweis, 732)

Note 6. Ibid., 380:

I do not consider that I am laboring under any illusion . . . that those individuals who have borne on the Second Clinic were also preceded by a conception. Then how comes the difference in mortality in the two divisions?"

Note 7. Skoda, for example, stated in his lecture before the Academy of Sciences:

"exudation at the wound on the inner surface of the uterus at the placental insertion site; or—less commonly—a partial or total conversion of the contents of a single or all the veins of the uterus to pus, with previous or subsequent exudation from the vein walls; or, finally, an exudation on the peritoneum."

Note 8. Loudon (2000, 96) wrote:

It had already become clear to his critics that the cadaveric theory as the explanation of all and every case of puerperal fever was untenable. In most cases of puerperal fever [. . .] it was rare for the birth attendants to have come straight to the patient from performing an autopsy. This was the major reason why Semmelweis's theory seemed wrong in general, even if it was correct under the special circumstances prevailing in the Vienna Lying-in Hospital. Semmelweis's first response was to postulate that the two cases mentioned above [a reference to the woman with carcinoma of the uterus who was admitted to the labor ward in October, 1847, and to the woman with a knee infection who was admitted in November, 1847 (see below). . .] must have produced dead matter (cadaveric particles) as a result of their lesions. But he soon abandoned the view that all cases of puerperal fever were due to 'cadaveric particles' or 'morbid matter' and substituted the important new concept of 'decomposing animal organic matter'.

Note 9. Semmelweis (1941, 581-582: "there are three sources from which the decomposed matter which causes puerperal fever is derived: every cadaver, every diseased person who engenders a decomposed matter, and all animal organic structures which have undergone decomposition'); ibid., 603: "Of the three sources from which the decomposed matter which causes childbed fever is derived, Scanzoni of course knows only the one: i.e. the cadaver . . . ; ibid., 645: "The reader knows that there are three sources from which the decomposed matter is derived . . ."; ibid., 677: "one of the three sources from which is derived the decomposed matter, which, introduced into the patient from without, causes puerperal fever, is indeed the cadaver."

Note 10. It should be noted that Tulodziecki, like Loudon (*see* supra note 2), inverted the chronology of what she called Versions 2 and 3 of Semmelweis's theory for Semmelweis realized that the causative agent could come from living persons with purulent discharges by December, 1847 (Tulodziecki's Version 3), and he first reported that the causative agent could arise from the mother's own lower

genital tract during his lecture to the Viennese Society of Physicians on 15 May 1850 (Tulodziecki's Version 2).

Note 11. Und wenn Sie, Herr Hofrath, trotz alledem mir eine Lehre untersrhieben, welche alle Puerperal-Fieberfälle durch Infectionen vom Cadaver entstehen lässt; so ist das entweder absichtliche Entstellung einer Lehre, oder es ist Mangel des Verständnisses meiner Lehre.

Note 12. It is conceivable that Semmelweis did as well, and that his statement about further animal experiments being superfluous may have been implicitly a response to Levy, otherwise his statements about further animal experiments, made immediately after he had quoted Brücke's memorandum, would be non-sequiturs as Brücke stated that he believed animal experiments were not the way to resolve any remaining doubts about Semmelweis's theory.

Note 13. Simpson . . . in gänzlicher Verkennung der Semmelweis'schen Lehre, welche er mit der englischen Doctrin von eineme einzigen spedcifischen Kontagium für identisch hielt, behauptete er, Semmelweis bringe nichts Neues und Alles sei schon längst in England bekannt. Nichtsdestoweniger veröffentliche derselbe Gelehrte später, offenbar unter dem Einfluss der Semmelweis'schen Mittheilungen, zwei Aufsätze, in welchen die Aetiologie des Puerperalfiebers fast ganz in Sinne von Semmelweiss aufgefasst und ebenfalls jene Krankheit mit der Pyämie (surgical fever) zusammengestellt wird. Man hat sehr mit Unrecht Simpson als den Ersten genant, welcher die Wöchnrin als Verwundete und das Wochenbettfiebert als accidentlle Wundkranheit bezeichnet habe. Dies ist zuerst von Cruveilhier und dann in sehr prägnanter Weise von Semmelweis geschehen.

Note 14. Oakeshott (1962, 162): "Usually, we interpret these past happenings in relation to ourselves and our current activities. We read the past backwards from the present…we call upon the past to speak to us in utterances related to the present: and what appears is a practical past.

Note 15. Lesky (1964), 46 §e.

"Dass seitdem Erlöschen der letzten Kindbettepidemie die Sterblichkeit eine geringere ist, daraus geht noch keineswegs hervor, dass dieselbe schon das Resultat der Chlorwashungen sei, dass somit die Ursache der Kindbettepidemie gerunden und der Wiedekehr derselben vorgebeugt sei. Dazu ist das Experiment noch zu jung und desshalb die Aneignung eines so grossen Verdienstes von Seite des Dr. Semmelweis noch voreilig und unmassend. "

Note 16. Article by author, "What did Semmelweis's discovery about the nature and cause of childbed fever owe to the Vienna Medical School and Joseph Skoda?" *Wiener Medizinische Wochenschrift* (in press)

Note 17. dass seine Fachgenossen, durch Annahme seiner Lehre, nothwendig eine gewisse Schuld eingestehen mussten. Sie mussten sich sage: Du hast, wenn auch unwissentlich und nur folgend den Anschauungen Deiner Zeit, vielfach schwere Erkrankung und Tod Deiner Mitmeschen berbeigeführt und hast, wenistens betrifft dies den akademischen Lehrer un Schriftsteller, durch Verbreitung falscher Doctrinen, noch in viel höheren Grade zu solchen Unglückfallen Anlass gegeben.