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Historical Perspective

From the 1930 International Johannesburg Conference on Silicosis, to “Tables” of Occupational Diseases, France, 2000 Onward: A Comparative Reading

Catherine Cavalin^{1,2,3*}

Through the concept of “thought collectives” in particular, Ludwik Fleck was a pioneer in demonstrating how much scientific knowledge is inherently made up of social and historical material. In this article, I propose to follow a Fleckian path by comparing the proceedings of the 1930 International Labour Office Conference on silicosis in Johannesburg on the one hand, and on the other the content of the debates that took place in France in the 2000s to revise the “tables” of occupational diseases which define the compensation rules for salaried workers in the French general (as well as the farm) health insurance scheme. The text offers an analysis of the striking similarities between these two distant sources, pointing out particularly the repetitiveness of ignorance and knowledge, and the nature of what can be admitted as a body of “evidence” in medico-legal issues such as the definition and compensation of occupational diseases. Am. J. Ind. Med. 58:S59–S66, 2015. © 2015 Wiley Periodicals, Inc.

KEY WORDS: occupational diseases; silica; silicosis; dust diseases; Johannesburg; France; Ludwik Fleck; compensation

INTRODUCTION

This article offers a comparative analysis of the proceedings of the 1930 International Labour Office (ILO) Conference on silicosis held in Johannesburg (designated

“J30”) and of the French “tables” of occupational diseases¹ (TODs) related to exposure to crystalline silica and silicates in the 2000s (designated “F2000”). Comparing these sources raises some intriguing questions about the way scientific knowledge builds up or is rediscovered only to be forgotten again. Indeed, this article will show that the questions raised in 1930 in Johannesburg and in France some seventy years later are strikingly similar.

This observation has led me to follow in the footsteps of *Genesis and development of a scientific fact* by Ludwik Fleck [Fleck, 1979]. This book has played a key role (and almost a

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¹ Since the law of October 25, 1919, which established the medico-legal nature of occupational diseases in France, these “Tables” have contained the list of the diseases which have a recognized occupational origin, with conditions concerning the duration of exposure and the time-limit for claiming compensation.

legendary role: see Braunstein, 2003; Latour, 2008) in the development of the sociology of knowledge and history of science from the moment when Thomas Kuhn read it in 1950 and when Robert K. Merton commissioned its English translation in 1979. This text helps us to understand how much scientific facts are inherently social and historical facts. Put briefly, the comparison I propose in this text rests upon Fleck's idea that "epistemology without historical and comparative investigations is no more than an empty play on words or an epistemology of the imagination" [Fleck, 1979, p. 21].

The similarities that we are going to discover between J30 and F2000 do not lead me to a pessimistic standpoint. As a member of the ERC SILICOSIS² research team, my aim is directed at finding possible ways to better understand the complex mechanisms involved in the health effects of exposure to silica and inorganic particles, which is the opposite of what a pessimistic attitude would entail. Here, this work will be undertaken from a perspective combining history and sociology of science.

What is at stake here is not whether there is progress or not in scientific knowledge. Endeavoring not to distort what Fleck has taught us, I would like to shed light on the paths that scientific knowledge "in the making" take. As Bruno Latour [2008, pp. 253–254] reminds us, Fleck was indeed more interested in this dynamic than in science as already "established" and he avoided a retrospective viewpoint that would have derided the knowledge of the past just because it was "old." Fleck proposed a cross-breeding approach between history, sociology, and science (medicine and life sciences, as is also the case for us here) to better understand the nature of scientific facts and how they arise and develop through "thought collectives" [Fleck, 1979, p. 38]. By so doing [Latour, 2008, pp. 255–258], Fleck invented a form of social history of knowledge in which the social dimension does not limit or invalidate the scientific findings but rather makes them possible.

Via the joint reading of J30 and F2000, I propose to apply to silicosis what Fleck suggested about syphilis: "it is not possible to legitimize the 'existence' of syphilis in any other than a historical way" [Fleck, 1979, p. 23]. In fact, Fleck added that "it would be a gross mistake merely

to assert that the syphilis concept could not be attained without the consideration of particular historical connections," meaning that a thorough critical examination of the "existence" of such a disease also means we must "examine possible laws behind these connections and discover operative socio-cognitive forces" [Fleck, 1979, p. 23].

In my comparative study of J30 and F2000, I do not pretend either to implement as deep an inquiry as did Fleck himself on syphilis or to develop an equally well-documented analysis in all the disciplines involved, i.e., medicine and life sciences, history of science, and sociology of knowledge. However, by questioning the scientific and medical content of both sources, I shall endeavor to highlight some relevant features that we can remember as framing ones in the "thought collective" [Fleck, 1979, p. 38] organized around silicosis throughout the 20th century. Moreover, by focusing on the scientific content itself, Fleck's approach is fundamentally different from a critical sociological analysis of knowledge. But as it does not exclude this critical view, I will also, secondarily, pay some attention to elements of the social context of J30 and F2000. My final aim will consist in making possible the continuation of a fresh cognitive approach to this disease today.

I will first justify the relevance of a comparison between these two sources that are far removed in time from each other. I will then highlight the similarities between the two historical moments of debate on silica and silicosis and help understand them through two phenomena: the difficulty of measuring true life conditions and the built-in social nature of scientific knowledge. At this point, I will explain that the cyclical nature of knowledge and ignorance does not prevent us from proposing new paths to redefine and renew the nosological entity of silicosis today.

WHAT SHOULD WE COMPARE?

The Nature of the Documents

The first corpus consists of the proceedings of the conference held at Johannesburg on silicosis from August 13 to 27, 1930, the 758 pages of which are available online [ILO, 1930].³ This source comprises two parts: the proceedings strictly speaking and the reports that the experts had prepared before the meeting, the content of which was discussed during the conference itself.

² The SILICOSIS research project is the product of a European Research Council (ERC) Advanced Grant, Paul-André Rosental being its principal investigator. It is located at the Centre for European Studies, Sciences Po, Paris. It aims at assessing to what extent inorganic particles and particularly crystalline silica dust may play a role (and which kind of role) in the triggering of a range of systemic idiopathic diseases such as autoimmune diseases (systemic lupus, systemic scleroderma, rheumatoid arthritis, etc.) and infiltrative lung pathologies (sarcoidosis, idiopathic lung fibrosis, pulmonary alveolar proteinosis, etc.). It is based on an interdisciplinary approach including history, sociology, survey research methods, several medical specialties, and mineralogy.

³ In the following pages, precise references to this text will be mentioned as [J30: p. X].

The second source, from the 2000s, has a functional unity but is scattered over various printed documents: the minutes of the debates which have produced the most recent versions of the TODs concerning exposure to silica and silicates in the French Social Security's General Regime (GR) and Farm Regime (FR);⁴ the experts' reports on the basis of which debates were conducted and decisions to revise the tables were made. These texts are all the more scattered as the ministries of Employment and Agriculture do not transfer them systematically to the French National Archives. Putting the whole corpus back together is a treasure hunt. I can reasonably hope that I have managed to gather nearly all the documents related to the latest revision of the GR's TOD and the main ones for the revision in the agricultural sector. As for previous revisions, the archives may have been definitively lost.

The French TODs are (re) negotiated in a system of joint governance between employers' organizations and workers' unions, under the decision-making responsibility of the relevant ministries' administration. The usual process takes place as follows. An ad hoc commission is set up to create or revise one or several TODs (within a permanent Commission for Occupational Diseases, COD). The commission examines experts' reports and, after generally two or three years of discussion, puts forward a new version of the TODs. This will usually be accepted by the administration and published through a decree, but it may also be rejected or modified by the administration, particularly if the latter receives some recommendations from the Council of State (the highest French administrative authority) or the Directorate of Social Security. This joint governance of occupational disease policy establishes the possibility of flat-rate compensation, subject to the criteria of duration of exposure and time limits for claims. This type of compensation implies a presumption of accountability, meaning that a worker has "only" to prove the duration of his exposure to a given risk and the period when this exposure took place in his career path.

A Comparative Reading

The proceedings of the Johannesburg conference which set out the definition of silicosis were meant to be foundational. Although not having this founding character and being less complete, F2000 presents some structural processes and characteristics which make the comparison possible and fruitful. Moreover, the long-lasting influence of J30 on the nosological and etiological boundaries of silicosis

for all of the 20th century has been well established by historical research [see Rosental, 2015].

In Johannesburg in 1930 as in France during the 2000s,⁵ the people who came together had in many respects the same assignment. All the participants had to (re) define silicosis as a pathological and social entity and to gather material on silica hazards, even though the context was different. Yet, despite this close parallel between the objectives of the meetings, the participants did not have the same social and professional background in the two cases.

In particular, the revisions of the French TODs institutionalize a negotiation between representatives of trade unions and employers' organizations under the responsibility of the administrative authority and on the basis of experts' technical reports. In Johannesburg, "lay" people (including members of the workers' unions) had a tiny part in discussions [Melling and Sellers, 2012, p. 118], contrary to the numerous "technical" experts, many of them closely linked to mining companies (occupational physicians as well as hygienists or even actuaries). Furthermore, at least one leading expert—Edgar L. Collis—was missing in Johannesburg, his invitation having been refused by the British Home Office and the Britain's Medical Research Council [Melling and Sellers, 2012, p. 117]. Collis was a representative of an "earlier generation of transnational experts and publicists" that had contributed to "[forge] a standard of objectivity conceived not just as technical but as explicitly social, drawing upon a distinctive contemporary vocabulary of social as well as medical science" [Melling and Sellers, 2012, p. 114]. In Johannesburg, he might have presented a thorough and alarming report on silica hazards, as the 1915 Milroy Lectures on silicosis [Collis, 1915a, b] and his decisive article on the (not only pulmonary) effects of silica in the human body (subsequent to the Johannesburg Conference [Collis and Yule, 1933]) enable us to understand. Consequently, J30's context was largely framed by technical, medical, and public health considerations as well as the preoccupations of South African gold mining companies, ousting most of the independent or overly critical "experts" (lay or academic).

As for F2000, the role and position of Claude Amoudru should be specially mentioned here: as the former chief physician for the Charbonnages de France (1970–1986), he was entrusted with the direction of the "work group on pneumoconioses" set up in 1999 to revise the GR's TODs concerning these diseases. This man who could thus be seen as the representative of the employers' medical views was (and still is) unanimously recognized as the person having the sharpest knowledge of health aspects of mining and pneumoconioses. When one peruses F2000, his invaluable

⁴ TOD n° 25 created by decree n° 2003-286 of March 28, 2003, (GR) and TOD n° 22 created by decree n° 2008-832 of August 22, 2008, (FR). The first French TODs related to silica, silicates, and silicosis were created respectively in these two social welfare regimens in 1945 and 1973.

⁵ 1999–2002 for the revision of the Social Security GR's TOD, 2008 for the revision in the FR.

contribution appears in his ability to make detailed and clear syntheses that helped workers' unions negotiate with employers' representatives.

On the whole, even in such debating contexts where the relative parts played by experts, laymen, representatives of employers, or trade unions differ, one common feature of J30 and F2000 is the capacity of regulatory experts to frame and shape knowledge on the disease.

SILICA AND OTHER KINDS OF DUSTS, SILICOSIS, AND OTHER PATHOLOGIES: AN UNSETTLING CONVERGENCE

Silicosis... and Another "Important" Disease (TB and Cancer)

Even though the carcinogenicity of silica was not a central preoccupation in J30 (the words "cancer" and "carcinoma" to designate primary site lung cancer were employed only 26 times in the 758 pages of the proceedings), the debates of J30 and F2000 present some similarities. They both deal with silica, silicosis, and another "important" disease: tuberculosis in 1930, cancer in the 2000s.

The relationship between silicosis and/or silica on the one hand and another disease (tuberculosis or cancer as two major public health issues) on the other hand was similarly investigated in J30 and F2000. Specifically, in dusty tissue, which immune mechanisms⁶ might be involved and could explain the progress of both diseases at the same time?

Contrary to the experts of the 2000s, the delegates of the 1930 conference did not think that silicosis could sow the seeds of primary lung cancer, but they pointed out that "this possibility must be kept in view" [J30, p. 243] and paid special attention to radioactive dusts [J30, p. 349].

In the 2000s, Prof. Pairon noted that the IARC's review of silica and cancer had to exclude numerous studies because of the airborne particle mix that one can find in most industrial environments and which rules out the identification of single causative agents for a disease such as lung cancer.

While we could have hoped that "evidence-based medicine" [Amiel, 2011; Haynes et al., 2006] would create a scientific consensus in the 2000s, this was not the case. This difficulty is illustrated by the results updated by the IARC since 1997 [IARC, 1997, 2012] on the carcinogenicity of crystalline silica. Pairon⁷ shows that the IARC's

results have been discussed over and over again,⁸ particularly the fact that the correlation between silicosis and lung cancer is stronger than the correlation between exposure to silica and lung cancer. According to which scientific criteria and for whom does the evidence need to be provided?

Other Dusts, Other Diseases – "Each Dust Calls for Special Study" [Collis, 1931]...

Two questions remain prominent today: does each type of particle provoke a specific disease? Are the mechanisms by which particles work in the human body of a mechanical [Loriga, J30, p. 48], chemical [Gardner, J30, p. 54], physical [Mavrogordato, J30, p. 41],⁹ or an immune nature?

In J30, Drs. Gardner, Russell, Simson, and Strachan were the main contributors regarding anatomopathological aspects. In the 2000s, the French experts focused rather on more general properties of chemical products, the biopersistence [Choudat, 2001, p. 3–4; Choudat 1999, p. 3; Amoudru, 1999a, p. 2] of mineral particles in the organism being for them the key notion, in accordance with the questions raised in 1930 about the solubility of silica [J30, pp. 23, 44–45, 60].

Are coal and silica still inseparable partners?

Given the importance of mining activities in J30, coal dust was as closely examined as silica dust. The terms in which the issues were debated in the 2000s are again strikingly similar [J30, p. 89; Choudat, 2001, p. 17–18]. "Pure" exposure to coal dust does not produce the same fibrotic effects on the lungs as exposure to silica dust. But actual working conditions being never pure, the etiological conditions a worker has been subjected to remain inextricable. The French GR's TODs set out a two-pronged arrangement: silica is separated from coal dusts by dedicating the TOD n° 91 to coal miners' COPD; in the TOD n° 25 devoted to the exposure to silica and silicates (including silicosis and coal worker's pneumoconiosis (CWP)), silica and coal are put together. But as Prof. Choudat noted, as the "clinical and radiological signs" [Choudat, 2001, pp. 7–8] of all the diseases involved in the

⁶ On this particular point, which is not the main aspect evoked by the delegates about the links between tuberculosis and silicosis, see Mavrogordato, [J30, p. 45]. Immune mechanisms later became central to analysis [Vigliani and Pernis, 1958].

⁷ Who himself took part in the experts' team for the 1997 IARC publication, as Claude Amoudru recalls [Amoudru, 2001, p. 2]. See [Pairon, 2001, p. 10; Pairon, 2007, p. 11].

⁸ Without speaking here of the agnotology kept alive by Eurosil, an association of European silica producers, which continues to contribute to the European Union's refusal to recognize the carcinogenic character of crystalline silica [Musu and Sapir, 2006]. The financial stakes linked to the recognition of the carcinogenicity of silica are considerable given the ubiquitous character of this chemical product.

⁹ Accumulation of particles in the case of silica; inflammation in the case of metallic particles.

TODs' revisions are often not specific,¹⁰ diagnosis is very often ambiguous.

Are silica and silicates clearly separated?

Although regretting a lack of studies, the 1930 congress delegates had little doubt that silicates¹¹ were involved in pulmonary fibrosis. F2000 reproduced the same drawbacks. For the first time “silicates” were distinguished as a proper entity in the GR's TOD n° 25 (and then in the FR's TOD n° 22) but paradoxically this did not mean that distinct etiological mechanisms had been identified. Choudat noted that the variable silica proportion in silicates is associated with a hotchpotch designated as “various pneumoconioses.” A nosological and etiological halo of silicosis thus persists.

By the Way, What Is... Silicosis?

The delegates of the 1930 conference were asked to reflect on the definitional limits of silicosis. The culmination of the final debates on the TOD n° 25 on January 17, 2002 is more surprising, with the definition of silicosis itself appearing to be neither clear nor consensual. As an agreement had just been reached to describe primary site lung cancer as “associated” with silicosis or a “complication” of silicosis, the social partners wondered how the diagnostic criteria of silicosis should be defined to make the compensation rules indisputable. Since anatomopathological examinations are hardly ever done, the definition of silicosis through “silicotic lesions” alone would have penalized many workers claiming compensation. Consequently, through social as much as medical criteria, silicosis was defined by “radiological signs or silicotic lesions.”

CAN EVIDENCE-BASED RISKS RELATED TO VARIOUS KINDS OF DUSTS BE ACHIEVED?

It could follow from the above that diseases caused by dusts will always lead us to the same questions, with no satisfying answers. I will try to understand why we seem to fail so often and

propose a positive reflection on the content of knowledge itself and on some precise obstacles it has to cope with.

How “Real Matter” Resists Our Efforts to Measure It

Measuring instruments and particle size

The delegates of the 1930 conference regretted the insufficiencies of available measuring instruments and the absence of a standardized international metrology, particularly as some of them underlined relevance of particle size [Badham, *J30*, p. 28; Spencer Lister, *J30*, p. 29]. Their questions on “ultra-microscopic” or “minute particles” appear very close to current investigations into nanoparticles.

We now have measuring instruments that were not available in 1930, but in F2000, the possibility of analyzing with electronic microscopic techniques the size of the mineral particles that can be obtained from bronchoalveolar lavage was never mentioned. Only two laboratories with an interest in both mineralopathological issues and environmental and occupational health possess these devices in France.

The difficulty of measuring the cumulative dose

In 1930, duration of exposure was one of the parameters for the calculation of cumulative doses [*J30*, 4th and 5th sittings], with the idea of testing what we would call today “threshold” and “dose-response” effects.

Encyclopaedic efforts are currently being made in some countries to compile data – duration of exposure among others. We find them in job-exposure matrices [Luce et al., 2006; Groupe de travail Matgéné, 2010; Delabre et al., 2010], ambitious data collection [Sauvé et al., 2013; Beaudry et al., 2013], and measurements intended to give realistic views of working conditions (e.g., the Colchic data bank in France).¹² However, exposure is still assessed via “the weight concentration in alveolar [i.e., respirable] dusts” [Vincent and Jeandel, 2000, p. 3]. While the particle size is highlighted as a critical parameter for environmental health [BEH, 2013], labor regulations go on establishing standards that are first and foremost based on a mass criterion.¹³

¹⁰ Hence also the failure in the revision of the TODs for COPD (n° 91 and n° 94) in 1999–2002, with the inability of the representatives of the employers and the workers to find an agreement.

¹¹ Free crystalline silica is silicon dioxide (SiO₂), the basic pattern of which is organized in tetrahedra of silicon and oxygen (SiO₄). Silicates are also mineral substances composed of SiO₄ tetrahedra, but between which metallic oxides are inserted (aluminium oxide, iron oxide, magnesium oxide, etc.). In this respect, asbestos is a fibrous hydrous silicate. When it is mentioned in the experts' debates in 1930 or in the 2000s, it is generally considered per se and not as a silicate among others. We can remember that crystalline silica and asbestos were part of the first French TOD dedicated to silicosis in 1945 in France. It was titled: “occupational silicosis” and subtitled: “diseases related to the inhalation of siliceous and asbestos-bearing dusts.”

¹² Measurements carried out by the laboratories of the regional Social Security units (CARSAT, Caisses d'assurance retraite et de la santé au travail).

¹³ Article R 4412-149 of the French Labour Code (modified by Decree n° 2012-746 of May 9, 2012) on the atmospheric concentration in chemical agents still defines the TLV in mg.m⁻³. Even though this mass criterion applies to respirable dust—which implies also the particle size, since “respirable” means that particles are small enough to enter the alveoli—an equal load of respirable particles measured in mg.m⁻³ can contain various numbers of particles according to their size. The more nanoparticles are present, the less the mass criterion reflects the physical reality of exposure.

Here again, lies a clue to the way regulatory norms and practices frame and shape knowledge about dust hazards.

Cohort studies: an imperfect tool to determine the latency period for the adverse health effects of particles

The 1930 delegates¹⁴ and the French medical experts in the 2000s repeatedly raised the question of the latency period of silicosis. The two main rapporteurs on this subject insisted that biopersistence of mineral particles appeared longer than was previously thought, justifying an extension of the time limits for claims in the TODs [Choudat, 2001, pp. 3; Pairon, 2007, pp. 3–4; COSMAP, 2007, p. 5].

While urging epidemiologists to develop cohort studies to assess this issue, Choudat has argued that even such cohorts cannot be a panacea because of various inherent methodological and practical limitations [Choudat, 2001, pp. 9–10]. And even with cohort studies, confounding variables may defy our efforts.

Overall, the quantification of exposure fails either for reasons linked to the nature of the task (true life vs experimental conditions), to our paradoxical oversights (the size of the airborne particles), and to the limitations of our best tools (insufficiency of cohort studies).

Scientific Issues: Explicitly Conceived as Social Issues by Experts

The history and sociology of knowledge and expertise have long highlighted the inherent social character of the production of knowledge, of observation itself [Daston and Lunbeck, 2011], discussing, for instance, the permeability of the boundary separating lay knowledge from professional or academic knowledge [Rabier, 2007; Callon et al., 2014]. Ludwik Fleck is one of the pioneers who drew our attention to the collective building of scientific knowledge. As for silicosis, the social character of knowledge and ignorance is closely linked to the intertwining of scientific and regulatory approaches, the latter being decisive in framing a number of the issues, as we have seen. The medico-legal character of occupational diseases in which inseparably medical (scientific) considerations are mixed with insurance (social) aspects parallels this “intertwining,” as has been well demonstrated [Rosner and Markowitz, 2006; Rosental, 2009; Devinck and Rosental, 2009]. And this “socio-scientific” cross-breeding plays a crucial role in the never ending re-discussion of the hazards caused by dusts.

The “statistics” of silicosis discussed at the Johannesburg conference

The social and economic aspects of the disease were a recurrent theme for the Johannesburg delegates, from the very opening of the conference [Irvine, *J30*, p. 24].

In particular, these two dimensions of silicosis underlay the subtitles of the eighth sitting: “statistics of silicosis” the real content of which was whether disease and work were compatible, and for how long in the miners’ lives. This social and political meaning¹⁵ was confirmed in the ninth sitting, more explicitly titled “legislation and compensation.” The medical and compensation questions were inseparable, with the expressed hope that the conference would help find ways to detect the disease as soon as possible while finding new ways of slowing its progression, i.e., to maintain a—sick but productive, productive even if sick—workforce underground [Irvine, *J30*, p. 25; Orenstein, *J30*, p. 81; du Toit, *J30*, p. 82].

“The definition itself of the disease, in order to define compensation, must be established in tandem between the social partners”¹⁶

Dominique Choudat expressed this opinion about coal and iron miners’ COPD to indicate that when social partners cannot come to an agreement on a qualitative diagnostic criterion, the absence of scientific consensus means the definition of a disease is no longer the responsibility of medical science. Cooperation is thus necessary to agree on social criteria.

This speech is consistent with the reflection of Amoudru [2001]. He explained that many miners suffering from CWP know they are compensated in the framework of the “silica TOD” which means that they are being recognized as “silicotics,” i.e., as having “the prestige of the silicotic man” or the equivalent of “the Legion of Honour of labour” [Amoudru, 2001, pp. 25–26]. As a result, (i) it is thus legitimate to adopt first a social (rather than a medical or scientific) standpoint to define occupational diseases; (ii) being “silicotic” is as much a social identity as a medical fact; (iii) compensation is (at least partly) organized according to symbolic criteria which are relevant for those insured.

¹⁴ See for example the “naïve” questions of Dr. Loriga on the latency period and how the onset of this progressive disease could be certified: [*J30*, p. 31].

¹⁵ Which is not a brand new revelation, since a critical tradition in the history of statistics has convinced us for a long time that statistics is never a flat and neutral description of reality (see for example: Desrosières [1993]; Schor [2009]).

¹⁶ Translated from: “La définition même de la maladie, en vue d’une reconnaissance, doit être établie en concertation par les partenaires sociaux” [Choudat, 2011, p. 57].

CONCLUSION: THINKING OF SILICOSIS AND DUST DISEASES AS POSSIBLY “EVIDENCE-BASED”

Do the strong similarities between the questions raised in 1930 and in the 2000s about silica and silicosis mean that science is condemned to wander almost endlessly? Or can we endeavor to renew research on silica and silicosis in some way, knowing that our definition of silicosis will not be “the” final one (which from a Fleckian perspective would have no meaning)?

Here, it seems important to highlight the potential contribution of anatomopathology, mineralogy, and immunology, both separately and in combination. These disciplines appeared to be central to the 1930 delegates’ preoccupations¹⁷ and surprisingly underestimated in more recent research or at least in contemporary regulations and rules of compensation. New means of investigation have developed that could allow us to identify and compare the nature and size of the inorganic particles involved not only in silicosis but also in some other granulomatous, inflammatory, and autoimmune diseases.¹⁸ For these diseases as well for pathologies suspected of being triggered by inorganic particles involved in air pollution, for instance, cytological, immunological, and histological mechanisms linked to the presence and circulation of dust in the body remain poorly understood. To keep on following Fleck’s lessons in our forthcoming research, we hope these are paths leading to a renewal of the “thought collective” not only built on silicosis but also on all the possible health effects of inorganic particles (including nanoparticles).

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¹⁷ For immunology, it would be more precise to say that the theme of immunity was very often associated with questions related to tuberculosis. But the notion of immunity was also raised regarding silicosis: was it true (and if so, by which immunological mechanisms could it be explained?) that there were places where workers were immune to silicosis?

¹⁸ J30 did not address these diseases as they were singled out mainly from the 1950s or 1960s onward, as shown by the bibliographical research conducted in the SILICOSIS project. See: Cavalin et al., [2013] or Rosental et al., [2013].

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