ACQUAINT PUBLICATIONS .....Disseminate your Research

Journal of Community Medicine and Public Health Reports ISSN: 2692-9899

Review article Volume 2 Issue 4

Validity of diseases' classification

Cem Turaman MD*, MSc

Free-lance public health consultant. Public health specialist, Epidemiologist, Tropicalist, Entomologist, Cankaya Ankara, Turkey

***Corresponding Author:** Cem Turaman, Free-lance public health consultant, Cankaya Ankara, Turkey

Received date: 25 May 2021; **Accepted date:** 01 June 2021; **Published date:** 05 June 2021

Citation: Turaman C, Validity of diseases' classification. J Comm Med and Pub Health Rep 2(4): https://doi.org/10.38207/jcmphr202100061

Abstract

The investigated health conditions have shifted from infectious conditions to chronic degenerative conditions within the last half-century. To adapt the disease control measures to the new situation, we had to adjust our definition of 'diseases of priority. However, this adjustment should have been done considering the necessary validity checks. Evolution on the one hand and regional diversity on the other, render the diseases variable entities. Therefore, a single, settled definition of diseases may not be reliable. Classification of diseases is necessary for their effective control if it fulfills the validity measures, thus all new definitions and classifications should be discussed in length, to set more effective disease control measures.

Introduction

Abdel Omran published his transition theory, with which we are familiar from its applications in demography and epidemiology, in 1971 **[1]**. The term indicates the transition from a relatively stable population with a mutually balanced high rate of mortality and birth rate into a population where mortality and birth rates are low due to the decrease in death rates first, then the birth rates. During this transition, acute morbidity, and mortality patterns were also replaced by the chronic morbidity and mortality models **[2]**. The transition period was long in northern countries which have accomplished to develop sooner, whereas in countries like Japan which have developed rather later, it took place in just a couple of decades, and in the southern hemisphere the transition has not been completed even though there is evidence that it has begun. Today even the developing countries, while still not being able to control the infectious diseases face non-infectious diseases or chronic or "non- communicable" health conditions **[2-4]**.

Demographic transition indicates the declining trend of the high mortality during the XVIIIth and XIXth centuries **[5, 6]** which primarily resulted from the decrease in deaths consequent to the living conditions and poverty **[7]**. However, even though the simultaneous disappearance of the black rat and plague at the end of XVIIth century in Europe remains a mystery **[8]**, the dramatic decrease in the mortality rates by the XXth century can be related to the stunning development of medical technology and antibiotics as well as the progression in the public health interventions and medical care **[9]**. Demographic transition is connected to epidemiologic transition through this channel: deaths, so far, have been the fundamental determinant of population dynamics in every era **[10, 11]**. In short, the transition was first due to development, then to public health interventions and improved health care **[12, 13]**. The common point of both cases is that the first ones to survive death due to transition were children **[14]**. Therefore, infant and child mortalities had been the most significant and sensitive health indicators in all the times, because of the relative vulnerability of thechildren, especially vis-a-vis the infectious diseases. But now?

Epidemiologic transition indicates the transition from epidemics which are transient but repetitive and unpredictable, rapidly spreading and retreating, into an era in which health conditions are permanent, slowly progressing, and long-lasting **[15]**. Thus, this is how those whose lives were prolonged thanks to the microscope, antibiotics and the improvement of hygiene got acquainted with long-term discomforts **[16]**. As a result, the rough mortality rates are no longer enough in defining human health; standardization of mortality rates, the recreation of the DALY and QALY estimations according to the requirements will not retard **[17]**.

To communicate or not to communicate, that is thequestion

Thus, because we have interiorized to ignore the "communicable" infectious diseases which are right outside of our windows as ameans of coping with them, we preferred to forget about them for a while **[18]**. However, we went black about plague pandemics **[8]**, leprosy was reserved for the poor regions of the World and massive leprosy epidemics became history **[19]** but the infectious diseases keep reminding themselves in various forms such as MDRTbc, SARS, and Ebola **[20-22]**. One reason for this continuity might be the overemphasis by WHO on non-communicable diseases which accelerated during the '80s despite the half a century-long failure of malaria eradication **[15]**. Even if this emphasis was based on a reliable prediction, it should not have underestimated the "communicable" infectious diseases **[16, 18]**.

More importantly, there is an odd aspect in this statement: what is

"communicable" is not the disease itself but its agent pathogen; similarly, offering someone a cigarette, familial eating habits, child abuse, and neglect are the agent pathogens of respective cancer, obesity, and behavioral disorders, and all three fit perfectly in the definition of "communicable". On the other hand, "non-communicable" exposure to vinyl chloride may result in a common source epidemic that leads to hepatic sarcoma **[23]**. Secondly, these two classes, namely "communicable" and "non-communicable" are not comparable in terms of their origins. The origin of the first is evolution, thus created by nature; the other is living conditions, thus created by humans.

Impact of evolution

Living pathogens evolved in a way that can be communicated from a seemingly healthy carrier or an ill host to a healthy host **[24]**. The pathogens have developed their communicableness characteristics in millions of years, during their co-evolution with their hosts **[25]**, so they have not been always communicable. Communicability of a pathogen shows extensive variability; it changes in time and can be communicated in different degrees in different places. This alone explains why communicability cannot be a universal criterion for disease classification.

For instance, the cholera epidemics which affected European capitals during the first half of the 1800s, caused a high fatality in ill hosts whereas, with the improvement of sanitation, the interest of the pathogen became dependent on its ability to adjust to avirulence which was not fatal for the host. Thus, the patients began to tolerate the pathogen which guaranteed the transmission of the disease agent from one person to another. The substitution of Bengal 0139 serotype coincides with this period and why it replaced the previous virulent isolate can be explained by sanitation improvement **[26]**. Another side-effect of sanitation measures is that they might have helped the emergence of "non-communicable" diseases **[27]**. The analysis of the development course of pathogen virulence mightbetter explain how evolutionary perspective can transform ascientific discipline **[24]**. Until very recently, it was deemed that natural selection would lead the pathogen and its host to a well- mannered co-existence **[28]**; however, the perspective of "why a pathogen would kill its nurturing host?" is completely wrong. The most important factor which forms virulence is its probability to communicate to a new host **[29]**; in other words, virulence forms according to the maximization of the pathogen transmission **[30]**. Before modern sanitation measures were taken, cholera patients could guarantee the continuity of pathogen transmission and infect other people; in this way organisms that led to severe diarrhea could be transmitted more **[31]**. This characteristic was selected during evolution because it maximized the communicableness of the pathogen. Plantation of clean water supplies prevented the transmission of the cholera vibrions from patients confined to bed to healthy people, therefore the selection advantage passed on to less virulent organisms **[32]**, thus the patients who could keep on wandering around even in their weakest states took on a vehicle role that transmitted the pathogen to potential hosts **[33]**. The degree of virulence might be affected by the rivalry of numerous pathogen isolates in the same host. In this case, the increasing virulence will be selected **[34]**. Vibrio cholerae holds the intestinal wall and causes diarrhea by excreting a toxin that triggers serous fluid secretion **[35]**. This characteristic of the Vibrio cholerae is evolutionally adaptive because it enables the transmission to other hosts **[29]**. We may assume that the selection functions in such a way that optimizes the net contagion between the hosts because it trades-off the low virulence (low contagiousness, however, the host lives long enough to infect other hosts) to the high virulence (high contagiousness, however, rapidly kills the host) **[34, 36]**.

Optimization of evolutionary the trade-off is especially important in sexually transmitted infections (STDs), because high morbidity decreases mating possibilities **[37]**. Therefore, we may assume that STDs follow a similar path with cholera. Infertility caused by STDs is a good example of evolutionary adaptation of the disease agents. An evolutionary perspective alone can help us understand how STD agents benefit from reducing the host's fertility and by which mechanisms the pathogenesis of these diseases acquire this feature **[37]**. Through their low mortality and morbidity, STDs' influence on fertility seems to be "target-oriented" and selective. Decrease in fertility and raise of pregnancy complication risks, together, may lead to alienation between the couples linked to not being able to reproduce, thus reduces the reproduction success of the host and transmitting itself to new hosts, the sexually transmitted agent the pathogen may be benefiting from the separation of the couples and increase in free sexual intercourse **[37]**. The birth of a child has a strong positive impact on the stability of a family **[38]**; on the contrary, infertility results in separation of the couples and alteration of the partners in most cases, and the rate of illegitimate sexual intercourse in couples without children are high **[39, 40]**. Therefore, the increase in peer alteration and free sexual intercourse is in the interest of an STD agent which can cause infertility or miscarriage; for growing free sexual intercourse catalyzes the transmission of the pathogen within the host population **[37]**.

So why the behavior of cancer which 'designs' thyself in a way that results in the death of its host is different? Is it different indeed? We do not expect the carcinogenic mutations and cancerogenic chemicals and mutagen physical agents to develop the disease in everyone just like we do not expect all the hosts infected by an infective pathogen to get sick. Even if we accept chemical and physical agents as communicable, how can we explain the communication of mutation? What about the genes? Do we notvertically communicate our mutated genes? **[41-43].**

Validity of diseases' classification

We must accept the fact that we have been insufficient in adapting to the health transition. One fundamental reason for this failure in the control of health conditions might be their definition and classification. The allegation that infections are communicable and new health conditions that are non-communicable might be a weak link in the control of diseases. Each definition includes areas that it over comprises and does not comprise; the definition roughly classifies health conditions in two as communicable and non-communicable risks to result in gaps in disease control. If we try to classify health conditions according to their different characteristics, we might propose these possible classifications: 1-According to their communicableness character: Communicable diseases/Non-communicable diseases; 2-According to the response of the host: Infectious diseases/Non-infectious diseases; 3-According to the duration of diseases: Acute diseases/Chronic diseases; 4-According to the result in the disease causes Degenerative diseases/Non-degenerative diseases; 5-According to the type of the pathogens: Biological diseases/Non-biological diseases; 6-According to the transmission of the agent: Directly transmitted diseases/Vector-borne diseases; 7-According to the source of diseases: Environment-issued diseases/Self-developed diseases. If the validity of each definition and classification is argued about a set of criteria such as relevance, applicability (feasibility), and acceptability, the strongand weak aspects of each classification could be seen more clearly. A relevant definition defines what should be defined. Relevance is the feature of representing only a particular class of the selected definition. Each definition must be appropriate to the field it is intended to be used for: Which classification and definition best meets the requirements of the priorities of the country including the politicians and the service providers, and the inhabitants? **[44]** Reliability, on the other hand, is the exclusive character of all the definitions within the same population under similar conditions. The definition we use should express the fact it refers to, specifying the differences and giving the same results loyally and accurately as far as the conditions remain intact **[45]**. Whereas, the classification function of a definition is designated with its distinguishing features, namely Sensitivity, and Specificity. Sensitivity is the distinguishing power of the disease classification of a given group of health conditions done without mistaking them with their similar ones. Specificity is the definitive character of a disease group with non-existing health conditions **[44]**. Another criterion, feasibility, is the utility of the information the classification provides and its practical usefulness. The basic constituents of the feasibility of classification are its acceptability for both the service providers and the service users, the duration of the preoccupation it causes, and its cost. Which classification is politically acceptable and can have the most stakeholder support? Along with these, criteria such as the austerity or the extra budget load of the classification, its efficiency, andeffects on health can be taken under consideration as well **[45]**. For instance, if we consider the first classification mentioned above which is currently used by WHO, "what do we communicate and not communicate?" would be a fair question. If the criteria mentioned above implicitly tackles this question: Disease is a case that evolves in the body of a living being and it is out of the question that this disease is communicated to the body of another living being as it is. What is communicated is the agent of the disease, not itself. Once it is communicated, what defines whether it will be hosted or not, meaning whether it will evolve into the disease or not, will be designated by the defensive ability of the new host. We can mention Dengue seroprevalence studies as an example **[46]**; within those who are exposed to the infection agent, only a mere part evolves the disease. On the other hand, during the 2000s Hamish McCallum and Anne-Maree Pierce have diagnosed a type of cancer amongst a little, endangered carnivore species in Tasmania which holds the skin and the mucous membranes that were communicable and spread only through communication. According to the scientists that examined this epidemic, it was "communicated" from one animal to another through inoculation with a bite **[47]**. Similar examples can sure be shown **[48]**; let us set aside these examples for now and focus on the definition of an epidemic. Two kinds of epidemics are known to epidemiology: epidemics caused by a common source and those which are transmitted from one individual to another **[49]**. Communicable disease epidemics resulting from a biological agent may occur in both ways. Influenza uses both ways perfectly while spreading, whereas HIV infection is defined as transmissible only from one individual to another. Though, an HIV positive individual may well play the role of the origin of a single-sourced epidemic by having unprotected sexual intercourse with more than one partner, by inviting to use his/her injector to several IV drug users and by donating blood. Likewise, are we to classify the Kaposi sarcoma which the HIV-infected people will develop as non-communicable? **[50]** To what extent the non-communicable disease patterns of an epidemic that are not related to a biological agent differ from the communicable ones? The cigarette offered by a friend may be the agent of Chronic Obstructive Pulmonary Disease, several cancers, and cardio vascular diseases; the delicious and fatty meals prepared by one's partner may be the cause of obesity and type-II diabetes; the efficiency methods imposed by the CEO which obliges the employees to sit in front of their desk all day long may cause osteoarticular conditions such as back pain in addition to the diseases listed above; and chronic stress may be the agent of some cancer types **[51]**, they all are "communicated" by "agent" individuals; those who communicate cigarettes, fatty food, sedentaryworking environments, and employer stress, all are communicable disease vectors. If I communicate the Coronavirus by sneezing onto the face of someone else in the coach, why wouldn't I communicate to my child the cancer agent by puffing out my cigars' smoke in his/her bedroom? So, what is the difference and how significant is the disease classification

used by WHO? These examples may be multiplied. If disease control measures target the patients alone but not those who communicate such disease agents and keeps on underestimating them, the control of such diseases will be doomed tobe insufficient.

Conclusion

The slogans of multilateral institutions that they believe to be applicable everywhere may become harmful agents even worse than both communicable and non-communicable diseases; acceptance without reflection may be the most malefic disease agent of the human mind. Review of definitions made in haste, by activation of implementations that focus on the underlying agents may be of great importance in the control of public health problems. Therefore, to exemplify, if we consider cancers as communicable diseases, public health interventions, drug research, and clinical trials would be targeting the real causes that are way beyond the apparent results.

To verbalise it as L.N. Tolstoi once did **[52]**, all valid classifications are alike; each invalid description is weak in its own way[1].

References

1. Omran AR (1971) The epidemiologic transition: a theory of the epidemiology of population change. Milbank Memorial Fund Quarterly. 49(4): 509-538.
2. Caldwell JC (2001) Population health in transition Bulletin of the World Health Organization, 79(2): 159-160
3. Olshansky SJ, Ault AB (1986) The fourth stage of the epidemiologic transition: the age of delayed degenerative diseases. Milbank Memorial Fund Quarterly. 64(3): 355-391.
4. Beaglehole R (1992) cardiovascular disease in developing countries. BMJ. 305(6863): 1170–1171.
5. McKeown T, Brown RG (1955) Medical evidence related to English population changes in the eighteenth century. Population Studies, 9(2):119-141.
6. McKeown T, Record RG (1962) Reasons for the decline in mortality in England and Wales during the nineteenth century. Population Studies. 16(2): 94-122.
7. Flinn MW (1974) The stabilisation of mortality in preindustrial Western Europe. J Eur Econ Hist. 3: 285–318.
8. Appleby AB (1980) The Disappearance of Plague: A Continuing Puzzle. The Economic History Review. 33(2): 161–173
9. Schofield R, Reher D, Bideau, A (1991) The Decline of Mortality in Europe, Oxford University Press.
10. Omran AR. Epidemiologic transition. In: Ross JA, Ed., International encyclopedia of population. London, The Free Press, 1982:172-183.
11. Barker DJ (1989) Rise and fall of Western diseases. Nature. 338(6214): 371–372.
12. Omran AR (1977) A century of epidemiologic transition in the United States. Prev Med. 6(1): 30–51.
13. Powles J (1992) Changes in disease patterns and related social trends. Soc Sci Med. 35(4): 377–387.
14. Caldwell P (1996) Child survival: physical vulnerability and resilience in adversity in the European past and the contemporary Third World. Social Science and Medicine. 43(5): 609-619.
15. Bloom DE, Cafiero ET, Jané-Llopis E, Abrahams-Gessel S, Bloom LR, et al. (2011) The global economic burden of non-communicable diseases. Geneva, Switzerland: World Economic Forum.
16. Rosenbaum L, Lamas D (2011) Facing a "Slow-Motion Disaster" — The UN Meeting on Noncommunicable Diseases. N Engl J Med 365(25): 2345-2348
17. Murray CJ, Vos T, Lozano R, Naghavi M, Flaxman AD, et al. (2012) Disability-adjusted life years (DALYs) for 291 diseases and injuries in 21 regions, 1990-2010: a systematic analysis for the Global Burden of Disease Study 2010. Lancet. 380(9859): 2197-223
18. WHO Library Cataloguing-in-Publication Data First WHOreport on neglected tropical diseases: working to overcome the global impact of neglected tropical diseases? World Health Organization 2010 ISBN 978 92 4 1564090
19. Jacobson RR, Krahenbuhl JL (1999) Leprosy. Lancet.353(9153): 655–660
20. Anonyme https://www.who.int/en/news-room/fact-sheets/detail/tuberculosis
21. https://www.who.int/csr/sars/en/
22. SteelFisher GK, Blendon RJ, Lasala-Blanco N (2015) Ebola in the United States — Public Reactions and Implications. N Engl J Med. 373(9): 789-791.
23. Heldaas S, Langård S, Andersen A (1984) Incidence of cancer among vinyl chloride and polyvinyl chloride workers. Occupational and Environmental Medicine 41(1): 25-30.
24. Morgan AD., Koskella B. 6 - Coevolution of Host and Pathogen, Michel Tibayrenc, (Ed.) Genetics and Evolution of Infectious Disease, Elsevier 2011: pp 147-171.
25. Armelagos GJ, Barnes KC, Lin J (1996) Disease in human evolution: the re-emergence of infectious disease in the third epidemiological transition. Anthro Notes. 18: 1–7.
26. Kavic SM, Frehm EJ, Segal AS (1999) Case studies in cholera: lessons in medical history and science Yale. J Biol Med 72(6): 393-408.
27. Okada H, Kuhn C, Feillet H, Bach JF (2010) The 'hygiene hypothesis' for autoimmune and allergic diseases: an update.Clin Exp Immunol. 160(1): 1–9
28. Haldane JBS (1949) Disease and evolution Ric. Sci.19(Suppl.): 68-76
29. Karlsson EK, Kwiatkowski DP, Sabeti PC (2014) Natural

selection and infectious disease in human populations. Nat Rev Genet. 15(6): 379–393.

30. Anderson RM, May RM (1982) Co-evolution of hosts and parasites. Parasitology. 85(2): 411–426
31. Snow J. On the Mode of Communication of Cholera, 8vo, London, 1849; 2nd ed. 1855
32. Seas C, Miranda J, Gil AI, Leon-Barua R, Patz J, et al. (2000) New insights on the emergence of cholera in Latin America during 1991: the Peruvian experience. Am J Trop Med Hyg. 62(4): 513-7
33. Bravo L, Ramirez M, Maestre JL, Llop A, Cabrera R, et al. (2000) [Toxigenic Vibrio cholerae non-01]. Rev Cubana Med Trop 52(2): 106-9
34. Kamble TK, More SR, Chavan SS, Kulkarni ND, Lodha NS, et al. (2000) Clinical profile of non-O1 strain-O139 of Vibrio cholerae in the region of Ambajogai, Maharashtra. J Assoc Physicians India 48(5): 505-6
35. Beubler E, Schuligoi R (2000) Mechanisms of cholera toxin-induced diarrhea Ann N Y Acad Sci. 915: 339-46
36. Singh DV, Matte MH, Matte GR, Jiang S, Sabeena F, et al. (2001) Molecular Analysis of Vibrio cholerae O1, O139, non- O1, and non-O139 Strains: Clonal Relationships between Clinical and Environmental Isolates. Appl Environ Microbiol. 67(2): 910-21
37. Apari P, de Sousa JD, Müller V (2014) Why SexuallyTransmitted Infections Tend to Cause Infertility: An Evolutionary Hypothesis. PLoS Pathog. 10(8): e1004111.
38. Anonyme. https://www.justice.gc.ca/eng/rp-pr/fl-lf/divorce/2004_6/p1.html. Accessed 15.02.2020
39. Ma L, Rizzi E, Turunen J (2019) Childlessness, sex composition of children, and divorce risks in China DEMOGRAPHIC RESEARCH. 41(26): 753-780.
40. Neal A., Groat H., Wicks J (1989) Attitudes about Having Children: A Study of 600 couples in the Early Years of Marriage. Journal of Marriage and Family. 51(2): 313-327.
41. Newman B, Austın MA, Lee M, King MC (1988) Inheritance of human breast cancer: Evidence for autosomal dominant transmission in high-risk families Proc. Nati. Acad. Sci USA. 85(9): 3044-3048
42. Anonyme. https://www.cancer.gov/about-cancer/causes-prevention/genetics. Accessed 14 Feb 2020
43. Gibson G. It takes a genome. New Jersey, Pearson Education Inc. Preface. 2009
44. Brinberg D, McGrath J.E. Validity and the Research Process. SAGE PUBLICATIONS Beverly Hills 1985: pp 19-20
45. Taylor CS. Validity and Validation Oxford University Press 2013: pp 10-20
46. L'Azou M, Assoukpa J, Fanouillere K, Plennevaux E, Bonaparte M, et al. (2018) Dengue seroprevalence: data from the clinical development of a tetravalent dengue vaccine in 14 countries (2005-2014). Trans R Soc Trop Med Hyg. 112(4): 158–168
47. Anonyme. Devil Facial Tumour Disease Senior Scientist's Scientific Forum 20 – 22 February 2007 Life Sciences Lecture Theatre, University of Tasmania, Sandy Bay Campus, Hobart.
48. Murgia C, Pritchard JK, Kim SY, Fassati A, Weiss RA (2006) Clonal origin, and evolution of a transmissible cancer. Cell. 126(3): 477–487.
49. Krieger N (2011) Epidemiology and the People's Health Theory and Context. Oxford University Press. 44(4): 1130–1132
50. Venkat Narayan KM, Ali MK, del Rio BC, Koplan JP, Curran J (2011) Global Non-Communicable Diseases — Lessons from the HIV–AIDS Experience. New England Journal of Medicine. 365(10): 876-8.
51. Sobhani ME, Molla AWM, Rahman MS (2010) A review on biomolecular basis of role of psychological stress in the development and progression of cancer. Mag Euro MedOnco 3: 136-142.
52. Tolstoi L. Anna Karenina. London, Penguin Books 2007: p:1