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## INTRODUCTION SPECIAL ISSUE 'THE FACES OF DEATH: REGIONAL DIFFERENTIATION IN CAUSE-SPECIFIC MORTALITY IN THE PAST'.

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### ABSTRACT

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In mortality research, much attention has been paid to the strong geographical differentials in mortality levels and in modern mortality decline, as the analysis of this geographical differentiation might hold the key to explaining the determinants of mortality change. The use of historical cause-specific mortality data has proved a challenging, although very insightful, means to this end. The four contributions to this Special Issue focus on cause-specific mortality in the past, both to reassess older data using new insights and to challenge existing insights by using new data and methods. These papers, of which earlier versions were presented during the 38<sup>th</sup> SSHA Meeting in Chicago, 21-24 November, 2013, explore mortality at different stages of the life course, ranging from early infancy to old age. Moreover, each paper revolves around a different group of causes of death. Although the papers are in many ways rather different, together they demonstrate how different data, theoretical frameworks and methodologies can push the boundaries of research into the trends and determinants of historical mortality patterns.

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KEYWORDS: MORTALITY, CAUSES OF DEATH, CAUSE OF DEATH CLASSIFICATION, HISTORICAL DEMOGRAPHY, SPATIAL DEMOGRAPHY

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Mortality has been referred to as 'the simplest' of the demographic processes (Hinde 1998, xi). In comparison to fertility, nuptiality and migration, it is conceptually and methodologically straightforward in that it happens to everyone and only once to each person. It is tempting, therefore, to assume that the products of research on mortality are also straightforward and not likely to be worthy of reassessment. However, it is a long known fact that death goes by a great variety of faces. Mortality rates fluctuate over time, but are also subject to strong national, regional and local differences. The vast spatial and temporal differentials in mortality and the modern rise of life expectancy have been a key issues in mortality research for decades. For most, the study of the strong variation of mortality through space and time is pivotal to an understanding of the determinants of mortality change. Despite considerable inquiry, however, the geographical differentiation of mortality remains an issue that is far less straightforward than the demographic event itself and the insights on the matter are as diverse as they are contradictory and inconclusive (Reher 2012, 121).

In this Special Issue, both highly contested and rather unchallenged theoretical notions regarding historical mortality research are addressed. It is well known that certain aspects of mortality, particularly causes of death, are trickier to interpret than others; debates which have been well rehearsed in Special Issues devoted to the subject in the *Journal of the History of Medicine, Continuity and Change* and *Historical Methods* (Alter and Carmichael 1996, 1997, 1999). By using epidemiological insights into the origin and course of the diseases and conditions that eventually caused death, the complex context determining mortality levels can be, to a certain extent, unveiled. Unfortunately, historical cause of death registers, however rich in data, reflect not only the new discoveries but also the shortcomings of contemporary medical science and civil registration. In recognition of some of these observations, efforts have been made to regroup causes of death to produce series which can be compared over time (see for instance: Bernabeu Mestre et al. 2003, Vallin and Meslé 1996, Meslé 1999). For infectious diseases in particular, there has been a general feeling that it is safe to use specific, easily identifiable causes of death.

The study of causes of death has been at the heart of some of the most influential products of research on modern mortality decline, whereby epidemiological changes are connected to

contemporary social developments. Abdel Omran defined the epidemiological transition, which coincided with demographic, economic and technological transitions that transformed nations into modern, industrialised countries over the past centuries. In the course of this epidemiological transition, the devastating effects of infectious diseases on population growth gradually receded and morbidity patterns began to be dominated by degenerative and lifestyle-related, or 'man-made', conditions such as cancer and circulatory diseases. To Omran, explaining the epidemiological transition was secondary to defining it. He did, however, identify three major categories of disease determinants: eco-biological determinants such as changes in pathological agents, political and cultural determinants such as the standard of living, and medical and public health determinants such as curative medicine. According to Omran, western mortality decline had been largely defined by eco-biological and social-economic determinants. Preventive and curative medicine only played a minor role (Omran 1971, 2005). The work of epidemiologist Thomas McKeown largely corresponded with Omran's theorising. In a process of elimination, McKeown stated that the majority of population growth in England and Wales during the 19<sup>th</sup> century could be attributed to the improvement of economic conditions and, consequently, a rise of the standard of living, allowing for a better diet and improving food hygiene (McKeown and Record 1962). He maintained that although the exposure to some infections was reduced by the instalment of water purifications systems and closed sewerage, such improvements amounted to relatively small contribution to mortality decline and were hindered by deteriorating working and living conditions brought about by industrialisation, whereas better diet mitigated these negative effects because people became more resilient to infectious diseases (McKeown 1976). McKeown based his findings on the major contribution of a decreasing number of fatal cases of respiratory tuberculosis to the overall mortality decline. He considered this to be the main proof of a better standard of living (McKeown 1983).

Omran's and McKeown's contributions have been very influential up to the present. Nonetheless, their findings have been discussed, re-evaluated and, partly, dismissed. Both Omran and McKeown have been targeted because neither had an eye for the great regional differentials in cause-specific mortality levels and trends. The four contributions to this Special Issue, in one way or another, re-assess the received wisdom from Omran, McKeown and other leading works within the field of historical mortality research. Earlier versions of these papers were presented at the 38<sup>th</sup> Annual Meeting of the Social Science History Association in Chicago, 21-24 November, 2013. Although the papers are very different on several accounts, they all aim to explain geographical differentiation in cause-specific mortality. These papers not only challenge existing insights by using new data and methods, but existing data is used to attain new insights into mortality trends and differentials in the past. This introduction starts with a brief summary of each paper, and follows by highlighting the methodological and substantive issues raised by the authors.

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## THE PAPERS

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The first paper by Van den Boomen and Ekamper challenges the received wisdom among both contemporary observers and some historical demographers that infant mortality rose in Dutch Roman Catholics communities in the last quarter of the nineteenth century due to a widespread cessation of breast-feeding caused by increasing cultural prudishness and a refusal to accept modern medical knowledge on proper infant diet. The paper uses cause-specific infant mortality rates for a full geographic spread of both small and big municipalities and finds that while there was significant correlation between Roman Catholic municipalities and a rise in water and food borne infant mortality, religion was neither a primary nor a sufficient predictor of where mortality rose. The rises in food and water borne infectious diseases occurred throughout many, but not all, of the Roman Catholic municipalities, but were also considerable in many of the non-Catholic municipalities in other parts of the country. In fact, the infant mortality patterns over this period are better characterised as regional rather than denominational, and are likely to be more complicated than a simple uni-causal explanation might suggest. Van den Boomen and Ekamper suggest that there might

be different explanations for the general infant mortality improvement in the coastal regions and the north, including the decline of malaria, and for the worsening in the eastern and southern region, including economic recession and an increase in female labour participation, but that local circumstances will have altered the way these played out in particular places. Therefore, they suggest that infant mortality levels in the Netherlands were defined by a regionally determined complex of health determinants.

In their paper, Reid, Garrett, Dibben and Williamson re-examine the attribution of causes of death by doctors and other informants, with a particular focus on deaths due to 'old age'. Using both national cause of death series for Scotland and individual death registrations, the authors argue that a rise in death rates from degenerative diseases, namely cancers and cardiovascular diseases, between 1855 and 1949 is likely to be the product of increasingly accurate certification of cause of death, as deaths formerly attributed to 'old age' were given more precise causes. This conclusion is carefully backed up with an analysis of the certification practices of individual doctors in a Scottish town, with consideration of the increasing tendency to provide more than one cause and of the possible ways that clerks allocated deaths to categories in the general nosology. They point out that unlike nosological changes, which produce sudden breaks in cause of death series, changes in the way that individuals (both doctors and unqualified informants) record causes of death are far harder to detect, even when they are precipitated by a sudden edict or instruction.

The paper by Reid et al. raises the need to consider the probable make-up of ill-defined and unknown causes, and the fact that this make-up will differ by age-group, over time and quite probably also by place. Aside from these cautionary warnings, there are significant implications for both contested and uncontested theories of mortality change. Theories, such as that of Thomas McKeown, based on trends in particular causes of death should be treated with caution given the evidence that those trends might not be as robust as previously assumed. In particular, Omran's theory of epidemiologic transition should be used with caution. This paper does not cast any doubt on the change in the distribution of deaths from infectious to non-infectious causes, but it does strongly suggest that there is little evidence for a rise in the risk of degenerative causes of death, and argues that it is thus inadmissible to refer to them as 'man-made' causes, as is often done.

In the third paper, Hinde presents an examination of the sex differentials in phthisis, or pulmonary tuberculosis, mortality in England and Wales. Hinde focuses on eight small areas, selected for their specific economic and social environments, and shows that geographical mortality differentiation was, in a large part, shaped by mortality differentiation in phthisis. The results reveal that phthisis mortality disadvantaged females in some, but not all areas. Furthermore, whereas overall sex differentials between male and female mortality were small or even negative in areas with a female disadvantage, these differentials were large in those areas where phthisis mortality disadvantaged men. Hinde considers two separate hypotheses as an explanation for the specific sex differentials in phthisis mortality. Firstly, he assesses the 'bargaining-nutrition' hypothesis, which argues that phthisis was primarily a disease of young women who were made vulnerable because their weak bargaining position in the household compromised their nutritional status. Secondly, he explores the 'return migration' hypothesis, which states that labour migrants contracted the disease elsewhere and returned home once the disease ran its course, only to raise phthisis mortality levels in their place of origin.

In his paper, Hinde finds little evidence that the bargaining-nutrition hypothesis can account for sex differentials in pulmonary tuberculosis mortality. Although it might have had some influence in poor agricultural regions, return migration of women who had previously left the area to work elsewhere might have accounted for the elevated phthisis mortality levels amongst women. In other areas, male phthisis mortality might have exceeded that of females because of diseased men returning home. Moreover, the bargaining-nutrition account does not explain higher phthisis mortality risks for men in other areas. The analysis adds evidence against the link between nutrition and mortality, which is so relied upon by theorists such as McKeown, and strongly suggests looking at alternative explanations.

The final paper by Kippen and McCalman is rather different to the other papers. However, it is an important contribution as it introduces a new and innovative dataset, created using web-based crowd-sourcing techniques to build a dataset containing the life histories of a cohort of convicts before transportation, under sentence and after emancipation in Tasmania. This paper deploys event history analysis to examine the influence of certain characteristics of convicts on mortality before and after release. They find that certain aspects of the convicts' background had a lasting influence on their chance of death under and after sentence. Convicts born in industrial-urban areas had a higher risk of death, both before and after emancipation. However, behaviour and punishment of offences committed under sentence were of great importance shaping mortality risks. Both physical and psychological insult accumulation during penance, such as solitary confinement, had a lasting effect on life-chances. The paper is particularly exciting in the novelty of the dataset which allows the consideration (almost unheard of in historical demographic studies) of the influence of characteristics such as temperament and behaviour on health and mortality.

These four papers, while all addressing mortality in the 19<sup>th</sup> century, are very diverse in theme, data and methods. They relate to different countries, to different age groups, and different causes of death. However, together they illustrate the different ways that historical mortality research can shed new light on old issues. In the following paragraphs, these insights are grouped into four broad themes.

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#### DATA: NEW AND RE-EXAMINED

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The papers in this collection demonstrate the continued advances in the collection, re-use and development of new data. Kippen and McCalman follow the recent trend in gathering individual level data to create life histories, which has seen the development of large-scale individual level databases such as the Umea Demographic Database in Sweden, the Historical Sample of the Netherlands, the COR-sample for Belgium and many more. In common with these, their data set includes data on standard demographic events: births, deaths and marriages, but the cohort is defined in an unusual way, with convicts transported to Tasmania from various places in the British Isles. These individuals can be followed intensively through the penal process, with the provision of rich contextual material on disposition and behaviour, and have also been traced using web-based crowd-sourcing through a variety of other data sources to pin-point individuals in space and time and to discover trajectories of occupation, migration, and family formation.

This process makes the dataset path-breaking in a number of respects. The first is the scope and method of the data collection: crowd-sourcing itself is of course not a new method, and is well-established for data gathering in historical demography. The Cambridge Group for the History of Population and Social Structure could be said to have pioneered the method in the 1960s when it used volunteers from local history societies to gather information on births, deaths and marriages for 404 parishes (Wrigley and Schofield 1989), but the internet has made it possible for volunteers to be involved in searching and recording a far more varied range of information, including newspapers and other local sources. This is arguably a more fun and rewarding task for volunteers but also provides a much broader range of information in the final dataset. The inclusion of a wider range of sources than those which provide just standard demographic events makes the tracking of adult life-histories easier as observations for individuals before deaths pinpoint their location and characteristics, and this is rare for Anglophone countries where continuous registration is uncommon. The convict records themselves are almost unprecedented in recording information on attitudes and behaviour, such as conduct during sentence and receipt of punishments of various sorts, and this allows a more direct consideration of some of the more elusive possible influences on health and survivorship. Of course it is important to consider issues of bias, both in the original selection of convicts and the possibility of visibility bias, but with proper care there are plenty of questions which can be asked and answered by this data. The paper shows the potential for

ambitious linking of unusual sources and we hope that this will be an increasing avenue for research in the future.

Reid et al.'s paper also uses individual level data but in an unusual way. Rather than examining the risks of mortality, they use it to cast light on cause of death certification practices by examining the way that individual doctors classified deaths among the elderly over time. This exposes sharp discontinuities in the practices of individual doctors as well as persistent differences between doctors which together provide compelling evidence of a shift in attitudes towards the causes of death which is unlikely to be visible in any other way. The analysis serves as a reminder that although changes in nosology are often easily visible in cause of death series, as clerks en masse change their allocation of particular causes to broader categories, changes in the original allocation of causes of death by doctors or relatives, although of comparable or greater impact on trends in cause of death over time and between places, are much harder to detect and need imaginative analysis to be fully appreciated.

While much path-breaking research in mortality in recent years has exploited individual level datasets, the papers in this special issue also demonstrate that important new insights can be gleaned from aggregate statistics, both new and old. The paper by Hinde uses the dataset constructed for the *Atlas of Victorian mortality* by Woods and Shelton (1997). Although the latter extensively used this dataset to demonstrate the various levels and types of mortality differentiation in England and Wales, Hinde shows how old data can be used for new objectives. Van den Boomen and Ekamper make use of one of the few newly constructed nationwide datasets on an aggregate research level. The distinctive thing about both these papers is the level of aggregation, and this brings us to the next set of new insights, that of spatial demography and geographical scale.

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## SPATIAL DEMOGRAPHY AND GEOGRAPHICAL SCALE

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In the early twentieth century, when demography emerged as a discipline, most of the work in the field could be broadly termed 'spatial demography' in that it focussed on the analysis of geographically aggregated measures. The second half of the twentieth century saw a move away from such 'macro-demography', which focused on mean levels and the average person, towards 'micro-demography', which focussed on individuals as the agents of demographic change, fostered by the development of new individual level datasets and by a desire to avoid the ecological fallacy (Voss 2007). In studies at the aggregate level there is always a risk that variables on group characteristics are interpreted as being measures of individual characteristics that exist within that particular group, and therefore erroneous inferences are made (Piantadosi, Byar and Green 1988).

While there is still a focus on individual level analysis today, the emergence of techniques such as multi-level modelling has avoided both the exception fallacy, which is the error of exceptional cases being used to reach conclusions about groups of individuals, and the isolation fallacy, which is associated with the danger of individuals being studied in the absence of any community or regional influences on their behaviour. This has, to a large extent, enabled the re-instatement of local and regional factors as important influences on demographic behaviour. The emphasis on individual data as the data most preferred in demographic studies dangerously devalues the important insights macro-demography still has to offer. The products of macro- and micro-demography may be quite different, but in combination they can shed light on the individual, local, regional and national contexts that shaped demographic developments through the ages (Johansson 2000). The papers by Hinde and Van den Boomen and Ekamper demonstrate that aggregate level data still has a valuable role to play in historical mortality research.

In a perfect world, scholars of historical mortality might wish for individual level data-sets comprising the births, deaths, and social-economic characteristics of all people in a given country over a long time period. Comprising such datasets is not only very capital-intensive, but also very time-consuming. Historical sources have to be made machine readable and linking the separate life events can be very difficult. Moreover, even with progressive advances in computing power, such

datasets may be exceptionally large and unwieldy to analyse. Most individual level historical datasets therefore concentrate on complete coverage for small geographical areas or on samples, often drawn on the basis of names, for larger areas. Both these sorts of dataset can produce valuable conclusions, but are less good at explaining wider geographical variation because the relationships which hold within them may not be applicable to a broader area. Restriction to such sources also means the wealth of data collected and published for small areas is ignored. The First Law of Geography states that everything is related to everything else, but things in close proximity to each other are more related to each other than things that are not (Tobler 1970). In other words, both individuals and communities that live or lie close to each other tend to be more similar to each other than do individuals and communities that live or lie at great distance from one another. Small geographical areas are more likely to be culturally and social-economically homogenous; they allow the more random elements of individual behaviour (the individual variation which is not the product of systematic differences in culture, social-economic status or behaviour) to be aggregated out; and they generally provide data over a wider geographical and temporal spread.

The papers by Hinde and by Van den Boomen and Ekamper indicate that the use of small-scale units can strike the right balance between individual observations and national means. Van den Boomen and Ekamper use cause-specific infant mortality for all 1,121 municipalities in the Netherlands at the end of the 19<sup>th</sup> century. Previous research at the regional level had been laid the blame for high infant mortality at the door of Dutch Roman Catholics, a conclusion supported by some individual level studies but contradicted by others. The municipal level approach in this paper sheds light on this conundrum, demonstrating that the conclusions drawn from the regional studies were subject to the ecological fallacy, and smaller individual level studies could not be generalised across a broader area. In contrast, Hinde gradually moves away from a broad regional analysis to a directed comparison of eight clusters of Registration Districts, chosen from over the 600 in England and Wales to examine the influence of local social-economic conditions, the intra-household bargaining position of women, and return migration on phthisis mortality levels. Hinde argues that in order to understand regional patterns of phthisis mortality, we have to study limited selections of small-scale units to understand the local contexts that define the impact of and resilience against the disease. Both studies show that the scale at which things are measured is pivotal and that using small-scale units might be excellent way to supplement micro-level studies with research performed at a macro-level.

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## IMPORTANCE OF MIGRATION

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Migration is the poor sister of demography, often ignored because it is so hard to measure, and particularly so in historical research. However there is a growing realisation that it can have far-reaching impacts on other demographic processes and their measurement. The absence of temporary migrants can cause a mismatch between numerators and denominators in conventional analysis (Blaikie et al. 2005), while results can be distorted when longitudinal results are compared against cross-sectional characteristics (Bocquier et al. 2011; Reid et al. forthcoming). Dyson (2011) has shown that the process of rural-urban migration has been fundamental to the process of demographic transition. In different ways, and more or less explicitly, all the papers in this Special Issue touch on the importance of migration or the urban-rural difference on which it feeds.

The lasting effects of place of origin are shown by McCalman and Kippen's study, which demonstrates that convicts born in an urban-industrial centre were likely to die younger than those born in more salubrious environments. They do not speculate on the intermediate mechanisms for this, but it might be reasonable to suppose that poor housing, air quality, and a heavy disease load in childhood had lasting effects on health later in life. Van den Boomen and Ekamper speculate that such factors might explain some of the geographic differences between municipalities in their analysis, and Reid et al. indicate that different levels of medical provision between town and countryside could produce spurious differences in causes of death. The most explicit consideration of

the urban-rural and migration nexus, however, is provided by Hinde, who delivers convincing support for the theory that the return migration of young women as well as young men who had contracted tuberculosis while working in urban centres, where diseased and healthy people lived in close proximity to each other, was at the root of high tuberculosis mortality in many rural areas. This has explicit implications for the way we think about the risks of disease-transmission and the roles of underlying economic and household structures in disease vulnerability.

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### CHALLENGING THE RECEIVED WISDOM

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Three of these papers question the received wisdom of the work by McKeown, Omran and like-minded researchers. The papers by Van den Boomen and Ekamper, Hinde and Reid et al. all recognise that registration errors might have grave consequences for historical mortality research. This is, of course, not a new observation when historical cause-specific mortality data is used. However, Van den Boomen and Ekamper find that even the re-classification of historical nomenclatures according to the ICD cannot correct for the under- or over-registration in certain categories of causes of death. They provide support that changes in recording practices can produce large changes in causes of death over time, demonstrating a trade-off between water- and food-borne infectious diseases on the one hand and tuberculosis and debility on the other. This finding leaves us with two different issues. Firstly, Omran implied that the modernisation of a region was reflected by its epidemiological state; meaning that modernisation diminished the role of infectious diseases. Even if nosological and aetiological challenges of historical nomenclatures are met by meticulous re-grouping, we cannot know whether modernisation had indeed reduced infectious disease mortality or whether low levels in an area were just a reflection of poor registration practices. Secondly, the geographical differentiation in cause-specific mortality might be a by-product of those particular registration deviations.

In his paper, Hinde explores whether geographical differentials in phthisis mortality are the result of misreported cases, but maintains that only a minor part of phthisis mortality change could be attributed to different diagnostic practices. This paper demonstrates that return migration is a more plausible explanation for high female phthisis mortality in rural areas than the bargaining-nutrition argument, particularly because there is little evidence for the connection between pulmonary tuberculosis and nutritional status in the epidemiological literature. Not only does this suggest that phthisis transmission mainly happened in urban area, but it also contradicts the link between nutrition and mortality that is such an important corner-stone of McKeown's hypothesis that improving nutritional status was the root cause of secular mortality decline in the late 19<sup>th</sup> century, and as such it has immense importance.

The contribution by Reid et al. disputes McKeown's hypothesis in a different way, by maintaining caution against the assumption that causes of death can be straightforwardly compared over time and between places. The demonstration in their paper that the rise in the risk of death from degenerative diseases, namely cancers and cardio-vascular disease, can be explained by a decline in the risk of death from old age, and the suggestion that respiratory diseases (often symptomatic of an underlying cause) might be subject to similar problems makes any broad theory which hangs on changes in particular causes of death over time subject to question. The McKeown hypothesis has been hotly debated for many years, but Reid et al.'s paper is braver in taking issue with the theory of epidemiological transition. It is important to remember, however, that they do not dispute the transition when it is characterised as a change in the balance of causes of death. There is no doubt that a decline in infectious diseases left more people to die, when they eventually did from degenerative conditions. What they dispute is that there was a rise in the risk of death at any particular age from degenerative diseases: even in old age people had no higher risk of death from degenerative diseases in the 1940s than in the 1860s, so these should not be thought of as 'man-made' diseases but as the natural result of ageing bodies.

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## CONCLUSION: THE DIFFERENT FACES OF DEATH

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These papers, in many ways quite different, turn out to fit together quite nicely. Together they can be thought of as providing insight into the risks of mortality across the life-span. In totality, they cover infancy, with the most changeable (if not the major) cause of infant death in the nineteenth century, diarrhoeal disease; adulthood with uncontroversially the major cause of death among young adults, respiratory tuberculosis; and old age, with the insight that there was probably little change in the risk of degenerative diseases among the elderly. Together they demonstrate how different data and approaches can provide insight into different problems and provide impetus for the value of a continued variety of data sources and methods which continually push the boundaries of research into the trends and influences on historical mortality patterns, and they show there is still a great deal to debate.

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