Zimmermann, Martina. "The narrative of loss in a growing biomedical and literary marketplace of Alzheimer's disease." *The Diseased Brain and the Failing Mind: Dementia in Science, Medicine and Literature of the Long Twentieth Century*. London: Bloomsbury Academic, 2020. 87–108. *Bloomsbury Collections*. Web. 26 Apr. 2023. <<http://dx.doi.org/10.5040/9781350121836.ch-005>>.

Downloaded from Bloomsbury Collections, www.bloomsburycollections.com, 26 April 2023, 14:46 UTC.

Copyright © Martina Zimmermann 2020. Released under a CC BY-NC-ND licence (<https://creativecommons.org/licenses/by-nc-nd/3.0/>). You may share this work for non-commercial purposes only, provided you give attribution to the copyright holder and the publisher, and provide a link to the Creative Commons licence.

The narrative of loss in a growing biomedical and literary marketplace of Alzheimer's disease

Medico-scientific progress during the 1980s and 1990s continued to advance the idea that Alzheimer's disease was an organic condition of the brain. The neurosciences consolidated the turn-of-the-century concept of degeneration in the biomedical dementia discourse, dressing it up anew as neurodegeneration. What is termed the period of biomedicalization, in fact, relies on the same principles of investigation and focuses on the same neuropathological features as had been relevant to Alois Alzheimer and his immediate colleagues. The efforts of three medical scientists to translate, during the 1980s, the research papers pertaining to 'the early story of Alzheimer's disease' suggest to me that the research community itself appreciated this continuity.¹ Following the huge investment by the NIA in Alzheimer's disease, much of what had been research on dementia until the late 1970s turned into research on Alzheimer's disease from the 1980s onwards.² *The Alzheimer's Disease Standard Reference*, edited by the old-age psychiatrist Barry Reisberg in 1983, convincingly documents this continuity.

Emerging lines of biochemical and genetics research now influenced the characterization of the neuropathological features (especially cellular degeneration, plaques and tangles) that Kraepelin had established as central to the disease pattern, and this characterization further cemented the narrative of degeneration, decline and loss as it involved images of the broken down architecture of brain cells. Throughout the 1950s and 1960s, electron microscopic magnification enabled the identification of senile plaques and neurofibrillary tangles at a structural level much finer and deeper than light microscopes had allowed for at the end of the nineteenth century. During the 1980s, these discoveries spurred the biochemical and, slightly later, genetic characterization of these features. Jesse F. Ballenger has placed the leading studies into their

historical context, but I am inclined to disagree with his view that electron microscopy ‘transformed the familiar and unproblematic pathology of dementia into something new and strange’.³ Rather, I see this line of research as directly continuing the work of the 1930s and 1940s that described an organic condition of tissue loss and cellular degeneration based on microscopic observations. For example, Arne Brun’s contribution to the 1983 reference compendium on Alzheimer’s disease reads like a summary of this early-twentieth-century neuropathological work. Reviewing evidence on ‘widespread atrophy’, Brun underscored that special techniques bring out ‘numerous senile ... plaques (SPs) and neurofibrillary tangles ... neuronal loss ... and blurring of cortical cytoarchitecture ... Affected neurons often appear to be in stages of terminal degeneration ... The number of SPs is also reported to parallel the severity of dementia’.⁴ Medical scientists now set out to break these morphological features down into smaller and smaller units, pursuing a ‘neuromolecular style of thought’ based on the principle that mental states expressed themselves in neural processes that could be ‘anatomized at a molecular level’.⁵

I will thus explore how the ‘aggressive program’ on Alzheimer’s disease pursued by the NIA impacted on patient and illness perception throughout the 1980s and 1990s, as it accelerated research on the cause or causes of the disease and the identification of risk factors and their early detection.⁶ My concern here is neither to judge the validity of various disease hypotheses nor to give a comprehensive overview of the various lines of investigation that medical sciences opened during the 1980s. Rather, I will focus on developments in relation to initial biochemical investigations and genetics research. In salient research papers of the 1980s and their popular scientific echoes, I will trace, in the first two sections of this chapter, how concepts of Alzheimer’s disease as a neurochemical communication disorder and as an inevitable, because inherited condition deepened ideas of loss in relation to dementia. These developments, together with what David B. Morris called ‘genuine warfare against the biology of old age’, significantly influenced how Alzheimer’s disease increasingly became understood.⁷

As I chart these developments, I specifically address where patients found themselves on a sociocultural level in the 1990s. At that time, images of Alzheimer’s disease as a condition in old age prevailed in a growing marketplace – a marketplace of dementia accounts as much as for service users (by which I mean patients and their carers) – but emerging genetics research became engaged in identifying the cause of early-onset forms of dementia. This question is crucial because the 1990s saw a surge in popular science writing on Alzheimer’s disease,

which made scientific research all the more accessible to the general public. And, as I will address in the final section of this chapter, this public included a steadily growing number of caregivers, who, in their own writing, were negotiating their parents' forgetting at a time when memory became – as we have seen in the context of Bernlef's narrative – a key cultural and political concern.

Neurodegeneration: The biochemical narrative of lost molecules, pathways and communication

With biochemical investigations of the 1980s, Alzheimer's disease became linked to notions of dying neuronal networks. The growing silence and isolation noted in socio-medical and cultural descriptions of the patient in the 1960s found a biochemical echo in illustrations of dying neurons in the 1980s. These biochemical studies described the loss of specific molecules in the brain, termed 'neurotransmitters.' The emerging idea of a breakdown of cellular communication was paralleled by ever-finer descriptions of collapsing cellular architecture and networks. The language employed in these studies can be traced back to the nineteenth century.⁸ But communication between cells as such came under scientific scrutiny only during the twentieth century.⁹

During the 1800s, lesion theories had fuelled enquiries into possible links between bodily functions and specific brain areas. With the rise of the neurochemical sciences in the 1960s, this thinking expanded to the molecular level. Neuroscientists identified distinct brain areas as being dominated by specific molecules – neurotransmitters typical for that brain area and produced in a set of cells prevalent in that area. A decrease in the amount or activity of the neurotransmitter was taken as indicating cell death in that brain area. It also served as molecular explanation for the loss of function of that brain area and, in consequence, the symptomatology of a disease. Alzheimer's disease research focused on cholinergic cells which are so called because they use acetylcholine as their neurotransmitter. In their choice of language, the first research papers that identified cholinergic cell death as central to the pathology established Alzheimer's disease as specifically a neurochemical communication disorder.

The first biochemical studies related to this field of enquiry were published in the mid-1970s. They detected changes in molecular activity especially in two brain regions: the cerebral cortex (which harbours our ability to think and reason) and the hippocampus (which is fundamentally involved in memory and learning). The authors reported that especially cholinergic neurons were lost in

Alzheimer's disease and concluded that the condition was 'a cholinergic system failure'.¹⁰ In 1982, the American neurologist and psychiatrist Peter J. Whitehouse and colleagues gave further weight to this concept.¹¹ They located the loss of cholinergic neurons to a specific area at the bottom of the brain known as the nucleus basalis of Meynert (nbM). This is important, because the 'large neurons in the nbM project directly to the cerebral cortex [and] similar neurons ... project to the hippocampus' (1237), that is, they connect to higher brain areas via elongated nerve cells. These observations led the authors to formulate the 'cholinergic hypothesis': that cholinergic cell death could explain impaired 'memory processes' and an altered 'cognitive activity' (1238).

These observations can be read alongside Laura Otis's research on nineteenth-century networking metaphors. This can illuminate how scientists came to use the projection metaphor and how it advanced their grasp of the condition. Otis's notion that nineteenth-century scientists' 'images of social bonds affected the way they saw those in the body' is directly applicable to the projection and connectivity concept Whitehouse and colleagues established.¹² Their metaphor had been prepared in the socio-medical and – if we think, for example, of Bailey's Faith Gadny – literary realm during the 1960s. The concept of the lost network conveyed knowledge on two levels: the patient's loss of social integration and her cerebral dissolution. I would go further and suggest that these concepts provided the basis for a neuroscientific understanding of the patient's loss of identity, joining Otis in her conclusion that 'then, as now, the [networking] metaphor invited all who encountered it to rethink their identities'.¹³ Based on these new insights, cognitive disturbances were beginning to be linked to communication issues, as, for instance, in the language of neurologist David A. Drachman: 'Interruption of this neuron–neuron interaction may result in decreased connectivity, resulting in *irretrievable "atrophic" loss of information*'.¹⁴ In other words, the loss of neurons became equated with the loss of contents on a cognitive scale, a move quickly reflected in cultural images of dementia, for example, Michael Ignatieff's novel *Scar Tissue* (1993).¹⁵

Ignatieff's narrator links 'the circuits ... going down' to the gradual loss of his mother's articulation and her ability meaningfully to communicate with him. He observes: 'Every day I come in here and one more has gone. She could walk three months ago. She could eat her food three months ago. She could talk. Now the words are going' (160–1). From there, it is a short step for him to explain to himself that she 'has left her self behind' (161). For Bailey, dementia was a condition of lost identity because of lost social connectedness. For Ignatieff, it is a condition of lost identity because of the lost ability to convey intelligence. This

understanding emerges from the narrator's meeting with Moe, a patient with amyotrophic lateral sclerosis (ALS), a form of motor neuron disease. Usually a fast progressing terminal neurological disorder, it exclusively affects voluntary muscle control, including the ability to speak, but leaves mental acuity intact.¹⁶ Linked to a computerized system, Moe tells the narrator of wanting to remain conscious.¹⁷ The narrator appreciates that Moe wants 'to see everything, to feel everything, to hold on to his awareness of life until the very last moment; and to him this consciousness – whatever its desolation – is worth fighting for to the last instant' (138). Moe is fully aware, integrated, connected. His continued awareness is the cognitive triumph over the silence of the narrator's mother – her Alzheimer's disease.

I want to linger on this difference between Alzheimer's disease and ALS, because it may help us better appreciate the persistence of communication breakdown metaphors in the popular dementia discourse. The sharp contrast fashioned in Ignatieff's novel between ALS as an illness of the body and Alzheimer's disease as a condition of the mind has become commonplace in cultural notions of each condition. As late as 2004, Jonathan Weiner's popular scientific portrayal of ALS, *His Brother's Keeper: One Family's Journey to the Edge of Medicine*, builds around the sharp contrast between the mind-defined ALS patient Stephen Heywood and the author-narrator's mother with Alzheimer's disease. Similarly, novels such as Michelle Wildgen's *You're Not You* (2006) depict ALS patients as autonomous. Kate reminds her caregiver Bec of her role as amplifier rather than interpreter, averring, 'This is me ... You're not you right now ... Remember it's me talking.' Her ultimate intellectual autonomy and control are portrayed in her requesting Bec to omit calling for help as she suffocates and dies.¹⁸ Also in Hazel McHaffie's *Right to Die* (2008) a highly intellect-driven ALS patient ultimately devises his own assisted suicide, making close friends and family accomplices without their knowledge.

The neurochemical view of lost networks and communication alone cannot account for the medico-scientific import of loss into contemporary ideas of Alzheimer's disease: the question of intellectual control is essential. This will become evident from looking at narrative representations of Parkinson's disease. The neurochemical model of disrupted networks, pathways and projections directly applies to Parkinson's disease, which progresses at a rate, and afflicts a population, similar to that afflicted by Alzheimer's disease. Diagnosed with Parkinson's in 1989, Joel Havemann tells his story in *A Life Shaken: My Encounter with Parkinson's Disease* (2002).¹⁹ The senior editor of the *Los Angeles Times* describes how Parkinson's 'disrupts the brain's circuitry' (35) and

expands from brain to body. He acknowledges the 'relentless march' (113) of his illness, pictures it as a 'robber' (108, 158) and alludes to the prospect of 'slowly slid[ing] away' (113), as 'the disease's victories' force him gradually to give up his independence (104). These are the notions of decline and dispossession we know from the cultural Alzheimer's narrative. But most revealing is Havemann's awareness of the 'threat of dementia' (114), given that some forms of Parkinson's co-present with dementia. 'I count myself lucky', he writes, 'that my brand of Parkinson's disease is the least destructive kind. And I regard myself as luckier still that insufficient dopamine production in the substantia nigra affects mostly movement rather than mental faculties. The symptoms of many other chemical imbalances in the brain are mental' (44).²⁰ Alzheimer's disease is Havemann's first example, and two consecutive chapters starkly contrast his excelling in work with how his mother-in-law's 'mind had permanently deserted her' (69). In this he agrees with other Parkinson's patients, among them Sandi Gordon, who feels as if 'my body were moving in slow motion. My mind was racing ahead full speed'.²¹

It is a widespread feature of cultural representations of Parkinson's disease that it is explored as a condition of control and balance easily lost. Havemann is not the only instance of a writer reminding us of his continued effort to 'control' the disease (122, 150), to ensure he remains 'self-sufficient' (157). In Jonathan Franzen's *The Corrections* (2001), the tyrannical patriarch Alfred Lambert glides into the 'relief of irresponsibility'.²² Or take Yaron Zilberman's movie *A Late Quartet* (2012). When the string quartet's eldest member, Christopher Walken's Peter, has to retire from his role as grounding cellist due to Parkinson's disease, the ensemble comes close to dissolution; it is Peter's wisdom that saves the group's coherence. What emerges from these reflections is that identity and self were considered intact when enacted by an autonomous and meaningfully communicating person within a social network.

In summary of these comparative readings, as the twentieth century came to a close, Alzheimer's disease was considered a condition whose cognitive component meant the patient's lost control and living a social death. Images we have come to know from socio-medical and cultural descriptions of the 1960s were bolstered by neurochemical insights during the 1980s. Soon they would be underpinned also by popular scientific descriptions, as I will demonstrate in one of the next paragraphs.

Thinking in terms of the trajectory of the disease discourse, it is crucial how firmly these concepts were grounded in the neuropathology of the condition. Whitehouse and colleagues reported findings in brains selected based on the

coincident detection of the very behavioural aspects and the pathological features defined by Alzheimer and propagated by Kraepelin: neurofibrillary tangles, senile plaques and degeneration of cells. I stress the continued interest in these features here, because the language that began to develop around them at this point further consolidated the concept of a neuronal communication disorder; it fed on, and further bolstered, concepts of system failure and network breakdown. This partly emerged from an understanding that the death of cholinergic cells was associated with neurofibrillary tangle formation.²³

By the mid-1960s, electron microscopy studies had described neurofibrillary tangles as bundles of filaments.²⁴ Scientists now wanted to understand what these filaments were made of and, in 1986, identified 'a normal brain cytoskeletal protein' (termed 'tau' or τ) as an important component.²⁵ The involvement and impairment of a cytoskeletal protein (a molecule stabilizing the architecture of a cell) in Alzheimer's disease appeared to explain, in structural as well as functional terms, Drachman's earlier idea of atrophic loss of information. According to the American neurologist Dennis Selkoe and his colleagues, abnormal tau in nerve cells could 'result in instability ... loss of effective transport of molecules and organelles, and, ultimately, neuronal death', an understanding formalized in the 'tau hypothesis' in 1991.²⁶ The representation of this hypothesis in popular science further solidified notions of Alzheimer's as a communication disorder. In *Decoding Darkness: The Search for the Genetic Causes of Alzheimer's Disease* (2000), for example, the neurogeneticist Rudolph E. Tanzi described the role of tau in terms that remind us of the language Otis identified in nineteenth-century scientific and social networking concepts:

In its normal soluble state, tau is essential to a cell's infrastructure. In essence, it forms the cross-beams of a cell-wide span of railway tracks – filaments called microtubules – which are constantly laid down and taken up for the purpose of shuttling types of protein cargo around a cell ... Corrupted tau might mean the kiss of death for a cell.²⁷

The epidemiologist David A. Snowdon went even further. He explicitly related these neuropathological features to social death, writing that the spread of tangles through different brain areas 'parallels, in part, the general pattern of the progressive loss of mental, physical, and social functioning that occurs in Alzheimer's patients'.²⁸ The further investigations of senile plaques, by comparison, eventually led to the evolution of the medico-scientific dementia discourse on a quite different level; but like neurochemical studies they further sharpened perceptions of the patient's loss and involution.

On genes and genealogy: The patient as specimen, carrier and type in research and popular science

By the mid-1970s, both light and electron microscopy-guided studies of plaques had revealed that they contained amyloid.²⁹ This insight led several laboratories to apply themselves to finding the molecular source (i.e. the precursor) of the amyloid-forming peptide. They did so in the conviction that amyloid beta was causing brain cell death in Alzheimer's disease.³⁰ This search soon centred on genetic investigations.³¹ Throughout the 1990s, these studies identified gene mutations that could either inevitably cause Alzheimer's disease or substantially increase the susceptibility to the disease. Amyloid beta played a central role in these studies. In the language of contemporary medical and popular science, it became the molecular culprit of the killer disease, which the NIA had placed under investigation fifteen years earlier. I believe that this depiction of amyloid largely explains what Margaret Lock described as the 'staying power' of the amyloid hypothesis in discourse developments; and it perpetuated, as I argue in this section, the narrative of Alzheimer's as a killer disease and the patient its victim.³² This narrative was further enhanced as genetic testing potentially transformed every individual into a classified entity.

During the late 1980s, the genetic information for amyloid beta was matched to the chromosome, which earlier work had identified as holding a defect causing familial Alzheimer's disease.³³ A chromosome is a packed structure in the nucleus of the cell that holds the genetic instructions of an organism, the DNA. A defect or mutation can alter or delete part of the information held on a chromosome and lead to pathological manifestations. For example, the amyloid precursor might suddenly be processed differently, yielding a form of amyloid (amyloid beta) that, instead of remaining soluble, deposits as plaques. In 1991, researchers around the British geneticist and molecular biologist John A. Hardy identified such a mutation in the DNA of subjects in whom early-onset Alzheimer's disease had been confirmed after autopsy.³⁴ These findings contributed to the formulation of the 'amyloid cascade hypothesis', which claimed that amyloid beta was 'the causative agent' of Alzheimer's disease.³⁵

The mass media response to Hardy's findings had already picked up on this: a headline in the *New York Times* in February 1991 promised that the causes of Alzheimer's disease would soon be found. The article told a narrative of genes doing 'their deadly work' and of 'sticky balls [of amyloid beta fragments] outside dead and dying nerve cells in the brain', closing on Selkoe's belief that 'blocking of amyloid will be a therapy'.³⁶ The popular scientific perspective on these

research endeavours amplified the idea of amyloid beta as the molecular version of Alzheimer's, the killer disease. For Tanzi, amyloid fragments were 'the toxic killer of neurons' (202). Most importantly, researchers themselves now adopted the figures of speech for this very molecule that the media previously had used for the disease as a whole: the amyloid precursor protein turned into a 'robber' (108).³⁷ The metabolic product resulting from a specific genetic alteration became the molecular surrogate of the condition itself. This development can be explained by the fact that, as José Van Dijck put it, the gene 'became reified as the central locus of vital activity, and molecules other than DNA were ... ranked as secondary'.³⁸ Genetics research thus appeared to be able to hunt down amyloid to the innermost core of the patient's body.

Geneticists themselves framed their investigations in this way. In the early 1990s, two further genes were identified – presenilin 1 and presenilin 2 – as they presented in a form of Alzheimer's disease with onset in the forties.³⁹ Selkoe introduced presenilin as a further 'guilty gene', which highlights how geneticists pitched their work in terms of 'a classic detective story' or an 'adventure plot' for the benefit of a general audience, on whose faith further public funding relied.⁴⁰ Rudolph E. Tanzi expanded on the researcher's 'detective work into Alzheimer's' (xvii) and the 'hunt' for genes and proteins (106, 130), and the neurologist Daniel A. Pollen described, in 1993, the ongoing work on presenilin 1 as 'the quest for the genetic origins of Alzheimer's disease' in the subtitle of *Hannah's Heirs*.⁴¹ Centrally involved in studies on inherited forms of Alzheimer's disease during the 1990s, Pollen described the family tree and the autopsy information of Hannah's descendants as 'a map for deciphering one of the genetic defects of familial Alzheimer's disease' (133). But with respect to the meaning and desired effect of the map metaphor, Van Dijck observed that it suggests 'it is only a small step from ... locating genetic aberrations to correcting those diseases'.⁴² Indeed, in an engaging and generally accessible register, Pollen drew parallels between research on Huntington's disease (a familial, incurable neurological disorder that affects both motor function and cognition) and Alzheimer's disease, writing that 'in Huntington's disease, just as in familial Alzheimer's disease, many of the primary steps in seeking a cause and then a cure came from the efforts of the families actually involved' (120). With Bruno Latour, we should consider Pollen's popular science publication a functional part in the networking strategies with which investigators reach outside the laboratory to garner support for their research activities; a book like Pollen's would particularly target those who help in the 'belief and spread of the facts'.⁴³ As will become clear in one of the next paragraphs, Pollen's writing contributed to the public learning about genetics

research on Alzheimer's disease, and it fostered belief in the potential of this new research strand to identify the cause of the condition. Focused on these goals, it was perhaps unavoidable that such presentations also triggered expectations for a cure, which pharmaceutical research has, to the present day, not been able to meet.

Pollen distinguished between individuals 'who escaped the defective gene' (34) and those 'carrying the abnormal gene' (65). Such terminology reinforced the idea that Alzheimer's disease engulfed the entire body of the person who, in Tanzi's words, was therefore 'genetically doomed' (38). This notion is also explained by the effect an ever-deeper molecular gaze would have, if we follow Foucault's claim:

The gaze plunges into the space that it has given itself the task of traversing ... In anatomo-clinical experience, the medical eye must see the illness spread before it ... as it penetrates into the body, as it advances into its bulk, as it circumvents or lifts its masses, as it descends into its depth. Disease is no longer a bundle of characters disseminated here and there over the surface of the body ... it is the body itself that has become ill.⁴⁴

Further findings supported the idea that Alzheimer's disease pervaded the entire body. By the mid-1980s, amyloid peptide was found in blood vessels and cerebrospinal fluid, and researchers began to identify a relationship between pathological changes in the brain and metabolic events in the periphery that might mirror these changes. Serum, blood constituents and even skin cells appeared to grant access to the understanding of central nervous processes, in the form of biomarkers of and diagnostic tests for signs of its culprit causes. By 1994, predictive genetic testing for Alzheimer's disease became a possibility. But given that no treatment was (nor yet has become) available, such tests only produce uncertainties.⁴⁵

Charles P. Pierce's *Hard to Forget: An Alzheimer's Story* (2000) explores these feelings of uncertainty.⁴⁶ It reveals the consequences for identity and personhood of the fact that genetic knowledge about Alzheimer's disease 'outstrips' the availability of pharmacological tools.⁴⁷ The American writer gives an account of his 'family's story' – his father was diagnosed with Alzheimer's disease in 1985 and the father's four siblings developed it, too. Pierce's narrative is the search for medico-scientific evidence for inherited forms of Alzheimer's disease that would be of potential relevance to his own prognosis. The writer sees his own future as determined by paternal nature and nurture as well as the medico-scientific response to such determination:

I saw in my father my walking future. It stepped into every corner of my life, like the subtle spread of a cold fog. We're all a little wary of the ways in which we find ourselves becoming our parents ... I may move through the stages myself – a specimen, until I am a lower primate, until I am a brain down the hall. My father was a specimen. Then he was a lower primate. Then he was a brain down the hall, not far from Dan Pollen's lab. (97)

Pierce draws heavily on what he calls Pollen's 'definitive account of the search for the Alzheimer's gene on chromosome 14' (203). This search relied, as Pollen detailed, on brain tissue analysis and the securing of 'specimen[s]' (169) from descendants of the same family tree. Pierce's reference to a lower primate might remind us of post-Darwinian ideas of degeneration. But its connection to medico-scientific specimens essentially suggests concern with objectifying practices.⁴⁸ Subjected to procedures of measurement, the patient turns into what Hacking termed 'an object of knowledge', knowable in genetic terms.⁴⁹ Pierce eventually decides against being tested, concluding: 'I am a specimen ... For all practical purposes, for all the good it will ever do me, I already know all I need to know' (173). Having Alzheimer's disease cannot be reduced to a gene locus nor can it make an identity.⁵⁰

The genetic model drastically impinged on the patient's understanding of identity and individuality. This comes into sharp relief when we read Pierce's decision against Jean Baréma's in *The Test: Living in the Shadow of Huntington's Disease* (2005).⁵¹ Aware that Huntington's disease ran in his family, the journalist Baréma saw himself confronted with a 'time bomb ticking away that will inevitably explode' (14). He faced 'a fifty-fifty chance of becoming a vegetable within the next few years' (32) and of being treated like a 'regressing adult' (45), while his brother and sister 'are slowly dying' of the same condition (75). Baréma's rendering of Huntington's typifies many of the cultural images we relate to Alzheimer's disease. Both Baréma and Pierce write memoirs that belong to a new generation of illness narratives which, according to Anne Hunsaker Hawkins, move away from 'medical science as invincible in its march to eradicate disease'.⁵² Pierce foregrounds the conceptual impact the disease label has on his father's as well as his own social situation. Understanding this label in terms of 'three of the genes ... [that] are death sentences' (94) turns being tested for Alzheimer's disease into what Baréma describes as 'the life test. The death test' (96). As cures for Huntington's (as well as Alzheimer's) disease continue to be elusive, similar accounts persist to the present day, patients describing their diagnosis as a 'curse'.⁵³

Ethical concerns regarding genetic testing came to be considered even more pressing once researchers identified mutations other than those predicting inevitable early-onset forms of familial Alzheimer's disease. The risk of having inherited an increased susceptibility to developing the condition at a later age became a matter of concern, channelling attention again towards Alzheimer's as a disease of older people.⁵⁴ This development was facilitated by epidemiological research. Epidemiologists maintained that, next to genetic factors, high levels of mental and emotional stress as well as environmental depletion could present additional risk factors. The healthy ageing discourse that developed in the wake of this work pitched brain health as a life project, but it also sustained the view that Alzheimer's disease was failed ageing.

David A. Snowdon, in 1990, attracted significant funding from the NIA for a large-scale epidemiological study with nuns.⁵⁵ The study added to the body of work that reflected a growing preoccupation with cognitive performance towards the end of the century. Its central claim was that a high level of linguistic ability (in this study reflected in idea density and complexity in writing) in early life can buffer cognitive decline; that, in reverse, lower levels of linguistic ability early in life could predict 'poor cognitive function' later in life (528). Patients indirectly were made responsible for their own later disease. And this led to the false hope that prediction could be tied to prevention.

The Nun Study dominated epidemiological Alzheimer's disease research throughout the 1990s. But it was Snowdon's move, with *Aging with Grace: The Nun Study and the Science of Old Age: How We Can All Live Longer, Healthier and More Vital Lives* (2001), to offer a popular account that turned 'cognitive reserve' into what the general public had to appreciate as a powerful asset against developing Alzheimer's disease. In popular terms, having or not having 'brain reserve' determined the separating out of future patients from the future healthy population, because 'a stronger brain has more of a reserve' and is 'more efficient, with better processing capacity' (45). Inevitably, the reader would have to picture the brain of an Alzheimer's patient in 'lesser', 'weaker' and 'poorer' terms. In another project related to the Nun Study, Snowdon carried this polarization further. He extrapolated from observations related to Alzheimer's disease to the healthy ageing of those with 'positive emotions' (194), and his eventual emphasis on 'longevity and successful aging' (216) seemed to insinuate that Alzheimer's disease was a condition of lacking success earlier in life and of failed ageing.

Cognitive reserve dominated public health initiatives that promoted healthy cognitive ageing since the mid-2000s. These initiatives went hand in hand with

the promotion of brain health by the Alzheimer's Association, which in turn contributed to Alzheimer's disease becoming a major public health concern in the new century.⁵⁶ Concurrently, more and more research and popular scientific outputs concerned themselves with questions of lifestyle and diet as determining cognitive health in ageing. *The Myth of Alzheimer's: What You Aren't Being Told about Today's Most Dreaded Diagnosis* (2008) by Peter J. Whitehouse is perhaps the most prominent example. It encouraged readers to 'tak[e] control of their own brain aging' by building up a cognitive reserve. This take on cognitive fitness resonated with the goals and interests of a growing cognitive health industry, which advertised its tools as helping people to 'think faster, focus better, and remember more'.⁵⁷

This popular scientific perspective on cognitive reserve encouraged lifestyle considerations. But it did not fundamentally change the situation of those diagnosed and labelled with the disease. Once positively tested for specific risk factors, the prospective carriers of 'amyloid burden' were left with the fear of falling victim to an incurable disease, of becoming a burden to their family and of being seen as losing their self and identity.⁵⁸ Guidebooks like Whitehouse's lifted some of the stigma attached to memory loss in old age, because it challenged readers to think about brain ageing 'not as a disease, but as a life-long process fraught with challenges'.⁵⁹ But this change in perspective has been slow in coming and, to the present day, keeps shifting in incremental steps only. As recently as 2017, the director of the New York Memory and Healthy Aging Services, Gayatri Devi, made the case for dementia as continuous variations in cognitive ability. Embracing the concept of cognitive reserve, Devi argues that Alzheimer's disease is a 'spectrum disorder that presents with different symptoms, progresses differently, and responds differently to treatment, with different prognoses, for each person'. But confirming the slow change in wider cultural understanding, she laments that we have 'not yet learned to associate Alzheimer's disease with functioning individuals, although this is, in fact, the majority of patients'.⁶⁰ Concurrently, concern about the worldwide marketization of cognitive fitness is mounting, with Peter J. Whitehouse, for example, calling for 'a counter-narrative that reinforces broader multifactorial notions of brain health'.⁶¹ Such concerns are particularly valid, given that brain fitness technologies have further enhanced the 'ambiguous image' of the ageing brain as 'improvable and "plastic" but also inevitably in decline'.⁶²

The idea of the brain as plastic and the patient as functioning individual gradually seeped into the wider cultural discourse, with literary writing beginning to envision Alzheimer's disease as a spectrum disorder from around

the mid-to-late 1990s. I will attend to these developments more fully in Chapter 6 and show how fiction that featured dementia as a condition of continuous capabilities returned agency to the patient.

Death in Slow Motion: Past identities, lost plots and old age in caregiver life-writing

During a period marked by a significant rise in popular science writing, caregivers began to be faced with scientifically sanctioned, desolate descriptions of their family member's condition – as encountered in popular writings such as Tanzi's: 'Dying nerve cells. Snuffed synapses. Severed pathways. Fallen brain regions. Impaired mental and corresponding physical abilities' (75). In this section, I want to think more fully about the position of the patient in the stories of caregivers at the time of these medico-scientific developments. What alternative concepts could caregivers hold onto as neurochemistry and genetics sanctioned socio-medical and popular beliefs about the patient's social death? To do these questions justice, we must include another factor that increasingly influenced the cultural dementia narrative: the marketplace. I already mentioned that scientific research could not remain free of external influences on its practices. The competition for public money furthered scientific outreach, which, in turn, held out hopes to patients and caregivers that the condition's cause or causes would soon be identified. Concurrently, popular scientific speculations about cognitive reserve created a market for preventive interventions. The production of literature could not remain exempt from such external deformations. During the period of biomedicalization the number of caregiver memoirs rose quickly, serving a readership increasingly aware of the condition but also in need of readable plots and redemptive endings.

During the 1990s, memory turned into 'a cultural obsession of monumental proportions across the globe'.⁶³ Caregivers became 'archivists' like the French writer Annie Ernaux, or turned 'hunter' and 'detective', and set off on an 'inquiry [back to the past]' like the American writer Mary Gordon and her conational, sociologist Lillian B. Rubin.⁶⁴ That such a wealth of adult-child narratives concerned with the personal past as part of post-war history emerged, in both Europe and America, especially during the 1990s, is perhaps best put into words by Lisa Appignanesi. Born to Jewish parents in Poland and brought up in France and Canada, she deliberates on the 'politics of memory' as follows:

As we approach the millennium, it seems we are all preoccupied by memory. To construct a future, we need to unearth new narratives of the past. Pasts which have been buried by repressive regimes and left to fester or pasts transformed by Cold War politics; or simply pasts relegated to the limbo of latency because they were too painful to think about.⁶⁵

Appignanesi's *Losing the Dead: A Family Memoir* (1999) shares similarities with Linda Grant's *Remind Me Who I Am, Again* (1998). As the descendant of Jewish immigrants, the British writer searches for identities for herself and her mother, who is losing her memory to vascular dementia: 'in her brain resided the very last links with her generation.'⁶⁶ In keeping with the contemporary understanding of identity as intricately linked to the ability to narrate, Grant explicitly ties notions of an intact identity to an undamaged awareness of one's own past. She describes her mother as 'continuously inventing for *herself* (and the rest of the world) a coherent identity and daily history' (156; emphasis original). With reference to Grant's narrative, Nicola King observes that it is 'not only the *content* of memories, experiences and stories which construct a sense of identity'; rather, the self in these narratives also depends 'upon assumptions about the function and process of memory and the kind of access it gives us to the past'. In the absence of her mother's memory, Grant – together with other adult children whose narratives King analyses – tells 'two life stories, her own and her mother's, which modify and reinterpret each other.'⁶⁷ Grant brings this meaning of memory work for the identity of both the writer and the written-about to the fore, and she places it in relation to the post-war trauma that fiction writers had begun to work through over a decade earlier: 'the whole thing about memory is that it's not just one member of a family losing their memory. And for the Jewish community it's even more complex because while all cultures are to do with memory, none more so than the Jewish community in which everything is about what was' (269).

But if we were to 'collapse memory into trauma,' Andreas Huyssen cautions, we would mark memory 'too exclusively in terms of pain, suffering, and loss.'⁶⁸ We have seen this understanding of memory, for example, reflected in Bernlef's account: the trauma of the past is so much stronger than dementia-induced forgetting that would or could conceal this very trauma. Memory is comprehended as an archive in these narratives, and I here address how archival notions of memory in caregiver accounts influenced the dementia discourse. I show how stories of the patient's emptying memory played on two ideas: the narrative of what Drachman termed 'irretrievable "atrophic" loss of information,' and the patient as living dead.

Like other adult children, Eleanor Cooney carries out an 'archaeological job' in the search for the cause of her mother's Alzheimer's disease.⁶⁹ *Death in Slow Motion: A Memoir of a Daughter, Her Mother, and the Beast Called Alzheimer's* (2003) reminisces on the American novelist's 75-year-old mother, the writer Mary Durant. It relies heavily on the metaphorical language perpetuated since the NIA's first information campaigns. Reference to the illness as beast turns the patient into its victim exactly like the killer disease had done in the 1980s, and the book's reception and marketing plays on the same imagery, promising an 'account of a hopeless and heroic battle'. At the same time, Cooney's excruciating honesty about her burnout and stress-related alcohol and drug abuse anticipates adult-child narratives of the later 2000s. These adopted an activist tone in the caregiver's struggle to receive appropriate healthcare support, among them Andrea Gillies's *Keeper* (2009) and Sally Magnusson's *Where Memories Go: Why Dementia Changes Everything* (2014). Much of Cooney's account illustrates the socio-economic and healthcare situation, as it presented itself around the turn of the century. *Death in Slow Motion* is the outlet of Cooney's travails caring for her mother-turned-child (35). She knits together her struggles to find a suitable nursing home and her search for the cause of her mother's illness. But to ascertain the cause of Durant's illness, for Cooney, really means to identify the root cause of her own caregiver depression. Discovering the reasons for her burnout, in fact, would, in Cooney's eyes, justify why she betrays her mother in placing her in a nursing home (111).

From the outset, Cooney is convinced that the death of Durant's husband Mike 'was the cause of her mental deterioration' (2). Cooney stresses that Mike's 'mind was going in every direction' (166) in the process of deciding to undergo (eventually unsuccessful) life-saving surgery. She particularly underlines: 'His decision to go ahead with the surgery was based, finally, on a sensible, practical, hard look at the statistical chances ... that his condition would, untreated, lead to stroke, early senility, ignominious decline. In other words, living death' (166). Reading these deliberations in the context of Cooney's negotiation with her mother's dementia brings out just how reduced in free choice she believes Durant to be, how limited in mind in contrast to Mike's limitations in body. But as Cooney probes her theory of Mike's death as the cause of her mother's illness, she has to look farther into the past. Her narrative reads like a celebration of the powerhouse relationship between 'Mike and Mary', to whom she dedicates her memoir. The loss of this relationship again typifies Alzheimer's as a condition of lost integration and lost communication.

Durant wrote her 'masterpiece, [which] got rave reviews, won some prizes' (121), together with Mike, namely *On the Road with John James Audubon* (1980). Cooney's choice of language, when she reviews Durant's writing 'about Audubon's decline' (123), establishes continuity between the mother's past (as well as her meaningful writing in the past) and her present state. This need to create life-story continuity emerges from the experience of illness as biographical disruption.⁷⁰ For example, the title of Cooney's final chapter, 'Sunset, Children ...', corresponds to the final lines in her mother's book (124), on whose taste and sensitivity Cooney comments: 'She knew what fine writing that was, knew it was sheer inspiration to end their amazing saga with those two words' (124). Picking this quotation from Durant's text suggests that Cooney acknowledges her mother's own life as an 'amazing saga'; it also implies Cooney's awareness that she learned her trade from her nurturer (81). Durant's 'powerful gift for character development' (7) now falls to Cooney herself. She has to write for her mother, because Alzheimer's develops her mother's (as well as her own) character during the eighteen months they share together.

Against this perceived loss, the narrative's early chapters centre on Cooney's vivid recollections of Durant as a loving mother. But most of all, Cooney's book contains an appendix, a space separated entirely from the daughter's writing, for a short story she had discovered in 'a rich stash of [Durant's] lost writings' (214). In posthumously publishing for the mother, Cooney's writing becomes a tribute to her mother 'as the whole person and ferociously good writer that she was' (215). But as Cooney's book turns into a device for maintaining Durant's identity as a writer and former self, this act underscores Cooney's perception of the patient as a 'living dead' (140, 166), unable to narrate and communicate. Cooney herself confirms this reading, when explaining that, in this appended narrative, her mother 'comes back from the dead' (214), continuing: 'This is my mother. And if you don't believe that Alzheimer's is the equivalent of death, try living with it for a year and a half. It's worse than death. In the long interval before actual physical death, the afflicted person is insidiously replaced by an imposter' (215). The narrative's final chapter then depicts Cooney's perception of her mother as a living dead most acutely, when Cooney returns to Connecticut 'to empty [her] mother's house' (200). The chapter's content and metaphorical language come very close to Galsworthy's use of the house as image. Constance Rooke comments that 'the house represents the self in society and contains (through memory and memorabilia) the "furniture" of identity'.⁷¹ Cooney is determined not to 'throw away a single book' (211), because these books, together with

‘thousands of notes, typed or scribbled – research, ideas, references, inspirations, ideas, ideas, ideas’ (212), embody Durant’s former fullness of life.

Cooney honours her mother’s legacy. But the narrative arrangement matches what Barbara Ehrenreich several years later criticized as ‘retreat from the real drama and tragedy of human events.’⁷² Cooney articulates her story, as Kathlyn Conway would put it, from a ‘position of authority outside the actual experience.’⁷³ Looking back on her caregiving experience, she can write about her learning, while avoiding talk about her mother’s complete bodily and mental disintegration. She can offer a ‘culturally validated narrative of triumph over adversity’ – with a storyline that is, given the condition’s trajectory, not available to the patient.⁷⁴ This culture of survivor literature would make it harder and harder for patients to find their own voice and reposition themselves in this mainstream narrative. It is, in fact, truly significant that Cooney places the account of Durant’s actual final days in an epilogue. This textual separation symbolizes the narrative place and time where Durant, in Nancy Reagan’s words, has ‘gone where I couldn’t follow’ (232). There, Durant is seen to lead an existence without a past, as ‘she doesn’t mention Mike anymore ... Terribly sad, but merciful. For her, and for me’ (225). Forgetting has turned into a disease-inflicted kindness. Also Appignanesi’s observation that her mother increasingly ‘denied what she didn’t want to know or chose to forget’ hints at more redemptive notions of forgetting towards the end of the century, a perspective increasingly found in much later life-writing, including, for example, Dana Walrath’s *Aliceheimer’s: Alzheimer’s through the Looking Glass* (2016).⁷⁵ In the overwhelming awareness of societal ageing (and increased risk for everyone to fall ill with dementia), forgetting becomes redemptive. The kindness of forgetting would become a version of the narrative of loss that could appeal to a mass readership for whom Alzheimer’s disease had come to embody the death of the self.

Presenile dementia, while fiercely pushed in genetics research, failed to receive a broader literary reflection in its own right at this point in discourse development (this changes towards the end of the first decade of the new century, as I will show in Chapter 7). In *Death in Slow Motion*, presenile dementia only features in passing when Cooney becomes ‘intensely aware of [two women] who are not a hell of a lot older than I am. They’re in their fifties, and have early-onset Alzheimer’s’ (230). In spousal memoirs, however, presenile forms of dementia find fuller examination. These spousal accounts of early-onset patients published in the late 1990s draw on very similar forms and categories of representation as the ones just highlighted for late-onset dementia in Cooney’s book. This suggests

how dominant the cultural image of Alzheimer's disease as a condition of old age continued to be at the end of the century.

Margarita Retuerto Buades's husband, José Antonio Gómez-Bárcena, was 56 years old when diagnosed with Alzheimer's disease in 1996. The Spanish lawyer's account, *Mi vida junto a un enfermo de Alzheimer* (My life next to an Alzheimer's patient; 2003), was one of the earliest caregiver narratives written in Spanish and published in Spain.⁷⁶ It echoes much of the need to share the actual experience of caregiving found in early adult-child accounts like Rosalie Honel's *Journey with Grandpa*. But unlike these early adult-child accounts, Buades's narrative is less concerned with a chronological exposition of events. Instead, it focuses on specific aspects of the caregiving experience which all emphasize that Buades has 'lost [her] husband' (89). 'Visits and friends' (95), for example, while emphasizing again the patient's shrinking social network, laments how 'at the same rate as my husband's condition worsened, these visits became more and more and more spread out' (105). And in the chapter 'From wife to caregiver' (79), Buades confesses: 'I miss my husband' (81). These images are not dissimilar to the dimensions of loss that Wayne Booth identified as 'universally pertinent' in ageing, which I referred to in the introduction: the loss of friends and partners in life.⁷⁷ In addition, Buades compares her husband's behaviour to that of a child (e.g. 31, 66, 72), an image also taken up in a national newspaper feature.⁷⁸ These similarities to adult-child narratives bring out the impact of the caregiving experience in Buades's account: the spouse with dementia, much like the parent with dementia, is considered a child. This is significant for the fact that Ann Burack-Weiss has described a 'history of reciprocity, along with a sexual history' as separating 'the situation of partners from other family care relationships' in contexts other than dementia.⁷⁹

Most importantly, though, Buades's reliance on medico-scientific and popular accounts links José's condition to one of ageing. She references, among others, David A. Snowden's *Aging with Grace*, seeks authority through mentioning José's clinical documents and specifically refers to evidence for the organic, neurochemical basis of cognitive deficits. Influenced by Reisberg's work on age-associated cognitive decline, Buades places memory loss alongside the 'inevitable and normal consequence of ageing' (54). In addition, she brings out how the condition 'progresses much more aggressively in younger people' (65). All things considered, I read Buades's rendering of her husband's condition as a cultural confirmation of, and commentary on, Bradley T. Hyman's observation in relation to the genetic dementia research of the 1990s. Hyman states 'that genetic factors lead to quantitatively worse disease but not to a qualitatively

different pattern of brain involvement'.⁸⁰ Even though Buades writes about a middle-aged patient, the images she uses, and the concerns she has, overlap with those of adult-child caregivers, whose patients are elderly. Her strategy for surviving 'all those years at war with Alzheimer's is to think that my husband is dead' (17).

Incidentally, Buades's reference to John Bayley's *Iris* (1998), in which Bayley writes about his wife's illness when she was in her seventies, is also significant for her perception of José as old.⁸¹ For while I do not suggest that Bayley's account gave her ideas about how to write about José, the sociocultural reach of Bayley's account made it a welcome source of comparison for caregivers across various cultural contexts and traditions, and this dominance helped strengthen the narrative of the patient as old, dependent and childlike.⁸² Rudolph E. Tanzi's reference to Bayley's account as giving the reader 'a sense of an Alzheimer patient's terribly limited world' (74), in turn, indicates a growing oscillation of language and images between the literary and popular scientific realm, and points to the central role literary writing assumes in amplifying a particular version of a narrative.

Bayley's text and the dementia narrative it portrays rose to extraordinary prominence in part because of its fast adaptation for the big screen in 2001.⁸³ Bayley starkly contrasts the couple's life together in the narrative's 166-page-long first part, 'Then', to a diary-like, forty-page rendering of the 'Now'. This textual arrangement underlines the Oxford don's perception of loss and provides the basis for the film's key narrative device. Richard Eyre's movie uses extended flashbacks to make most tangible emotional and physical disruption as well as discontinuity; it does not offer a 'life-course perspective'.⁸⁴ Rather, in constantly switching between the 'young' (Kate Winslet) and 'old' Iris (Judi Dench), the film contrasts Murdoch's fulfilled and youthful life as an intellectual and Bayley's struggle for her attention, with Murdoch's inability to continue to write in the present day of her old age and illness.⁸⁵

Bringing Alzheimer's disease to the big screen enhanced clinical (if not medico-scientific) appreciation of the patient's and caregiver's experience, also because the Alzheimer's Association influentially endorsed the film. We might read the positive reactions of geriatricians and psychiatrists to the film in the tradition of the geronto-psychiatric work on senility in the 1960s and 1970s.⁸⁶ But they were also the product of whole-person approaches to dementia care that began to emerge during the 1990s – developments that became instrumental in giving a voice to patients themselves.

As Alzheimer's disease became a household word during the 1980s, and wider societal attention grew for neurochemical and genetics research, the patient became increasingly marginalized. Pictured as suffering communication breakdown and objectified in caregiver accounts that zeroed in on the burdens of care, those who experienced the illness directly had been pushed ever farther away from the centre of attention. As mentioned previously, the writings of patients began to be published in 1989. However, it took another decade of growing patient organization, linked to a rise in psychosocial work on Alzheimer's disease, before patients would begin to be perceived (and perceive of themselves) as political agents. Agents who were able and authoritative enough to establish their own version of the illness – removed from the socio-medical, neurochemical and healthcare narratives of loss as well as caregivers' pessimistic accounts, amid a growing marketplace.

Patients were additionally pushed to develop a counter-discourse by a further evolution of the medico-scientific view of Alzheimer's disease and its related portrayal in the mass media. With the advent of *in vivo* imaging methodologies, illness progression could be 'seen' in the living brain. As Tanzi put it, for the community of medical scientists and clinical neurologists 'now to actually *see* direct evidence, to be able to actually watch the disease progress in a living person, is quite amazing. It gives us a way of tracking the rate and severity of the disease's progression; of knowing, for the sake of applying drugs, who will get the disease sooner than later' (245–6; emphasis original). Only there were no drugs available.