How do we make intelligible the abnormal psychological behaviors of human persons with dementia? Many contemporary clinicians and researchers recognize the need for integrative pluralistic and holistic approaches to understanding, treating, and caring for persons with dementia. The hylomorphic ontology that undergirds Aristotelian philosophical anthropology clearly presumes some form of integrative descriptive, explanatory and causal pluralism. My aim in this paper is not to defend these contentious claims but to presume them as my point of departure. I shall articulate a rough pencil sketch of how an Aristotelian philosophical anthropology provides resources for integrating the ontological, biological, psychological, and social factors relevant to making intelligible the actions of those suffering from dementia. I being and end with psychosocial factors, for it is here that our initial encounter and later caregiving of persons with dementia takes place. Our initial encounter will bring us to the biology of dementia, and from here we will briskly run through some ontological highlights concerning the way Aristotelian hylomorphism can integrate all these factors.

1. Psychosocial Factors of Dementia

Let us start with Gilbert Ryle’s celebrated distinction between “thin descriptions” and “thick descriptions,” which has been fruitfully developed by the cultural anthropologist Clifford Geertz. An example of a thin description is that of an eyelid closing and opening. This thin description of a phenomenon leaves out the variety of “thick descriptions” that also might be true of the same phenomenon. Imagine a fraternity of teenage boys. (1) One of them got some grapefruit pulp in his eye and now has an uncontrollable eye “twitch”. (2) A second boy, for some furtive reason, “winks” to give a covert signal to his friend. Now imagine a (3) third boy, perhaps an older sibling, who notices this “wink” and then “burlesques a wink” to parody and make fun of the second boy—his younger brother. Imagine further, that the second boy on an earlier day conspires with his friend to employ winks to give a signal. This second boy then goes home (4) to practice winking in the mirror; here he is not closing and opening his eyelid to wink and communicate a signal, he is “rehearsing a wink.” One and the same “thin description” is true of the all; they all closed and opened their eyelids. But if this was the entire account, the most important features of human significance would be left out. Dogs, cats, and horses can blink and twitch their eyes, but they are not known for winking with intrigue, parodying winks, or rehearsing them. Geertz rightly argues that these “thick descriptions” are essential for making human lives intelligible, that is, for making sense of the culturally shaped psychosocial facets of human life. It is because ordinary human life is intelligible in the ways revealed by true thick descriptions, that there can be theoretical disciplines like cultural anthropology, psychiatry, and cognitive neurology. We need “thin descriptions” too, but they are never enough to make sense of the prisms of significance that constitute meaningful human living and dying. Let us stipulate that the “thinnest” of descriptions depict mere physiology. The “thinnest” of descriptions therefore leave out even “smiles” and “frowns.” A “smile” is already a thick description and one of its correlative thin descriptions might be “the exposing of the teeth by the opening of the lips and the upward raising of the corners of the mouth.” As Alasdair MacIntyre has argued, the human body—like other animal bodies—is an expressive body that can express a panoply of embodied signs. It is therefore also an interpretable
body, that is, the observable living psychologically expressive body of a rational animal.\textsuperscript{vi} In the language of the ecological psychologist James Gibson, it is a body which, like many features of our environment, signifies a variety of observable biological, psychological, and social affordances. ‘The affordances of the environment are what it offers the animal, what it provides or furnishes, either for good or ill.’\textsuperscript{vii}  

Elizabeth Anscombe taught us that any human behavior admits of a multitude of true (thin and thick) descriptions of what a human is doing. Some of these true (thick) descriptions articulate what a human is doing intentionally, and some describe other kinds of human behavior, like what persons are doing unintentionally and nonintentionally, i.e., against one’s reasons and without any reasons, respectively.\textsuperscript{viii} Anscombe famously argues that “What distinguishes actions which are intentional from those which are not … is that they are the actions to which a certain sense of the question ‘Why?’ is given application; the sense is of course that in which the answer, if positive, gives a reason for acting.”\textsuperscript{ix} Anscombe spends a considerable part of her book Intention explicating this account of intentional action. I note a few highlights for our purposes: Anscombe’s view does not require that all genuine intentional actions are presaged by some explicit verbalization to oneself or aloud either antecedently or after the performance. All that is required is that the intentional actor is in principle able to provide a sincere answer to the relevant question “Why?” that provides the actual reasons operative in one’s psychological behavior. And because, for Aristotelians like Anscombe, the bodily behavior of animals is largely observable psychological behavior, we can observe the embodied practical reasoning that constitutes the observable intentional activities of other human beings without their explicit articulation of what they are doing. We can observe the intentional action of a man running to catch a bus or a thief pickpocketing that man on the bus or bets taken with a head nod during a Balinese cockfight as much as we can observe a smile or a frown.\textsuperscript{x} These “thick descriptions” are among the many observable—even if learned and defeasible—psychological, social, and cultural affordances we take for granted in our everyday living.

MacIntyre has shown us just how much is taken for granted and rarely made explicit—and rarely needs to be made explicit—in our everyday abilities to make sense of the intentional and non-intentional behaviors of others and ourselves. Human actions only make sense against the background of our understanding of various routines, institutions, practices, traditions, and the different kinds narratives that render each intelligible. The mere thick description of a man running to catch a bus presumes a shared understanding of the routines of getting around a city (e.g., home to work), of institutions of public transportation, and of narratives or scripts about, for instance, the dependability of public transit in some cities or of certain individuals to be on time for scheduled activities. To make an action intelligible—either our actions or those of another—requires that we are able to place it within the settings and sequences of an ongoing narrative.\textsuperscript{xi}  

It is often the recurrence of our inability to make intelligible certain actions of our family or friends that moves us to seek professional help. Imagine a married couple of 45 years; the husband is now 70 years old and the wife 65. Over the past three years the wife has only gradually come to notice some significant and worrying changes in her husband’s behavior. At first she interpreted these abnormal behaviors of her husband as typical signs of aging or his frustrations with retirement. They seemed to fit a typical narrative of getting older. Over time, however, her husband’s behavioral changes became even more pronounced. Not only was her husband becoming profoundly more forgetful, he also seemed less sensitive to the concerns and feelings of others. He started making rash decisions and gave bad or unintelligible explanations for these actions—or, he simply denied he did anything of the sort. She spoke with her children who also noticed some of these changes and they agreed he should see a clinician. It is because his actions became unintelligible to his wife and children—and seemingly to himself as well—that medical advice was sought.

Notice that psychiatrists or neurologists are sought in order to help explain the person’s abnormal behavior, not their ordinary behavior. They are not sought to make intelligible either a person’s intentional actions or even their ordinary non-intentional behaviors because both of these
kinds of actions can be placed within the ordinary healthy norms, routines, practices, and forms of life that make intelligible both non-intentional and intentional action. Sub-psychological biological factors are always essentially relevant conditions for all human behavior because we are rational animals, but they do not make thick descriptions of ordinary intentional and non-intentional behavior any more intelligible qua forms of psychological behavior. Sub-psychological biological factors are only needed to make intelligible a very limited sphere of psychological level human non-intentional behavior, namely, those abnormal, refractory, or pathological forms of human non-intentional behavior that do not fit into the narratives of healthy psychosocial behavior.

Many clinicians start their diagnostic process with a detailed interview of the patient with a close family member. The patient is asked to articulate a detailed narrative history that focuses especially on the last three years when he or his family first started noticing the changes in his behavior. His wife is invited to note any relevant gaps she notices in her husband’s narrative and the clinician is interpreting this complex clinical conversation against the backdrop of thousands of case studies, diagnostic profiles and criteria, her own clinical experiences, and a great deal of her own clinical hermeneutical prudence. She recognizes the ways the elderly gentleman’s wife—often unintentionally—assists or co-enables her husband’s efforts to answer questions and communicate his recent life history. The wife does this with her slight nods of the head, facial reactions and other subtle gestures that appear to confirm or query her husband’s responses. At some points in the interview the wife draws attention to a number of inconsistencies or peculiarities in her husband’s story or profound changes that he has not noticed. She mentions that he has quit bathing daily and no longer changes his clothes. At first, her husband is surprised to hear this description of his behavior. “Do I really do that?” He asks. He then quickly confabulates the following justification: “I guess I quit showering and changing my clothes because they were no longer dirty.” When asked, “why did your clothes and body quit getting dirty all of the sudden?” he just gets confused and throws up his hands saying “they just did!” The clinician will recognize certain signs and patterns connected to the specific stereotyped kinds of perseverating behaviors and memory loss in her patient. For instance, he recently started a new ritual of “taking all of his books from their shelves in order to re-stack them. He sometimes did this several times a day and he would swear quite violently (which he would not have done before) if any of the books were disturbed.” The clinician will recognize the ways these behavioral symptoms are unlike the kinds of deficits typical of Alzheimer’s patients. For instance, this patient does not seem to have deficiencies in day to day memory; he has started to disappear and go on walks without any purpose and without considering the need to tell his wife, but he always finds his way back home. More telling is his apparent lack of insight or awareness that he is performing any unusual behaviors; he just does not seem to notice what he is doing or has just done. This apparent anosognosia (i.e., deficit in self-awareness or lack of insight into one’s illness) has been a source of frustration to his family in the face of his sometimes insensitive remarks and unusual impulsive and rash behaviors. He also seems not to notice his insouciant lack of motivation yet without exhibiting any signs of sadness or depression. The clinician also knows to ask about changes in hygiene (like those mentioned) and diet; families will often be surprised to discover there might be some connection between these abnormal behaviors, refractory unintelligible actions, and the husband’s recent voracious cravings for sweet and fatty foods without any prudential inhibition and inconsiderate table manners.

The hermeneutical work of the clinician relies fundamentally on these detailed, often co-narrated, ongoing personal histories and thick descriptions of her patient’s behavior. It is on the basis of this narrative understanding of her patient’s case that she, against the hermeneutical background of her expertise and healthcare policies, makes judicious interpretations and prudential recommendations about how to proceed. Given her expertise and the salient markers exhibited by the patient in his personal story and in his telling of that narrative, she judges this definitely appears to be a case of dementia. Julian Hughes characterizes dementia as:

an acquired, chronic, usually progressive disorder, which affects multiple parts of the brain, and can lead to symptoms and signs that involve higher cognitive functions, activities of daily living, emotional and social behaviors, and physical abilities. 

- 3/14 -
There are a number of forms of dementia including Alzheimer’s disease, vascular dementia, dementia with Lewy bodies, frontotemporal dementia, and other less common forms. Alzheimer’s disease is the archetypal form of dementia and is the most prevalent, about 50% of all cases of dementia. (See Appendix) Our clinician is beginning to suspect her patient is likely suffering from a case of frontotemporal dementia (FTD), and probably not Alzheimer’s disease because his everyday memory is intact and it is probably not dementia with Lewy bodies (DLB) for the same reason and he does not show any sign of having hallucinations.

The clinician decides to have her patient come in for a battery of neuropsychological tests; this time the testing will be done alone. What researchers on aging, memory loss, and dementia have come to realize are the important ways in which, especially elderly married couples, function as tightly ordered co-operative social-units living within highly structured and stable environments, routines, practices, traditions, scripts, and narratives. These are unquestionable goods, but they can keep concealed the invidious developments of certain diseases like the various forms of dementia. After the death of one of their elderly parents, children sometimes discover soon afterwards, much to their surprise, that their surviving parent is actually in an advanced state of dementia that had not been noticed—even by children close to their parents. This is because of the way the recently deceased parent provided subtle but extraordinary forms of co-operative support to the surviving spouse. By conducting these neuropsychological tests in the absence of close family and friends, the clinician is able to temporarily take away from their patient the kinds of social supports, affordances, and scaffolding they might be relying upon and which can conceal their disease. They also help clinicians to rule out possible other explanations for various cognitive impairments. These tests often include language, memory, and some drawing tests. For instance, they might ask the patient to remember three random words—e.g., Barn, Alligator, Umbrella. Next they are asked to draw the face of a clock, and then to draw the hands indicating a specific time (e.g., “10 minutes after 11” or 11:10). After drawing the clock the patient is asked to repeat the three words. The clinician can compare the patient’s results in these kinds of tests with expected norms for measuring normal and abnormal “higher cognitive functions.” Some clinicians are beginning to include more affective and social testing to balance this focus on “higher-cognitive tests” which exclusively focus on language, memory, and executive functions. Other clinicians have also voiced concerns over clinical diagnostic practices that focus too much or exclusively on what Steven Sabat and Julian Hughes call “defectology.” We need tests like these, but they also need to be administered in a humane way.

What this narrative history combined with these neuropsychological tests provides the clinician is an intelligible profile of her patient’s psychosocial competencies and deficits that can be compared with her background understanding of various psychiatric exemplars. The ways she conducts the interview and tests requires a distinctive kind of clinical hermeneutical prudence. She selectively asks questions and carefully interprets the embodied gestural and linguistic responses. What she is trying to do is engage in a dialogue with her patient, not to draw out some philosophical error or contradiction, but to elicit distinctive stereotyped psychosocial behaviors—both intentional and non-intentional ones—that clinicians recognize to be affordances or signs of distinct pathologies. The entire diagnostic process is one that is saturated by “thick descriptions” of the spectrum of psychosocial abilities and disabilities that the clinician is able to bring into the light through her interview and tests.

2. Some Biological Factors of Dementia

So far I have briskly surveyed some of the psychosocial factors related to the diagnosis of dementia. At this point, the clinician is probably beginning to form a preliminary diagnosis of her patient as being in the early stages of frontotemporal dementia (FTD). In addition to more
neuropsychological tests, interviews, and vigilant observation of the developing symptoms of her patient, she is likely to turn to various biological tests which require the use of specialized technologies, like MRI, EEG, blood tests, and genetic analysis. For instance, if the cerebral spinal fluid from a lumbar puncture of the patient contains high concentrations of the tau protein and if an MRI reveals significant bilateral atrophy in the frontal lobes and/or anterior temporal lobes, these—among other biological markers—will help narrow down a diagnosis of frontotemporal dementia; perhaps it is the behavioral variant of frontal temporal dementia (bvFTD).xix Sometimes these biological tests overturn preliminary diagnoses made on the basis of interviews and neuropsychological tests, but they can only do so because of the way thick descriptions of MRI or genetics are bound up with thick descriptions of alternative biopsychosocial disease exemplars.

Unfortunately the causes of FTD are not known, but one biological marker is the excessive accumulation of certain kinds of proteins within brain cells (e.g., the tau protein) which leads to neurodegeneration and neuronal cell death. This results in significant atrophy of these parts of the brain. The loss of these areas of the brain have deleterious effects on the patient’s abilities for practical reasoning, intentional behavior, controlled emotions, and linguistic communication. There is no cure for FTD and the life expectancy is about six years, though some have lived upwards of twenty years. The patient might be prescribed antidepressants, like selective serotonin reuptake inhibitors (SSRIs) to help with behavioral symptoms.xii The medications used to treat Alzheimer’s—e.g., Acetylcholinesterase (AChE) inhibitors and Memantine—are not effective for FTD, and unfortunately, far too many FTD patients are misdiagnosed as having Alzheimer’s dementia.

3. Aristotelian Hylomorphism: Integrating Psychological and Subpsychological Levels

What can an Aristotelian philosophical anthropology add to this complex biological, psychological, and social picture of dementia? First, unlike most of its rivals from philosophy of mind, Aristotelians maintain that the intelligibility of human behavior in ordinary life and in the clinic would be unrecognizable and impossible if our observations had to rely entirely on “thin descriptions” of mere bodily movements. The problem of other minds—and all the additional problematic implications that follow from it—is not an Aristotelian problem because we reject the mental-physical dichotomy and its purported intuitions; for example, that the only observable facts we can observe are facts captured entirely by thin descriptions of mere bodily movements. Aristotelian philosophical anthropology aims to secure the facts about the observable intelligibility of human behavior and unite them with facts concerning human development in ethics, social interactions, and psychology and the ongoing discoveries in the biological sciences. These are contentious claims that I cannot defend here.xiii

Second, we need a metaphysical explanation for how all this hangs together, one that elucidates how there can be such a complex nexus of interactions among ethical, social, psychological, and biological factors related to ordinary and abnormal intentional and non-intentional human activities. Aristotelian hylomorphism claims it can provide this. However, it is important for Aristotelians not to exaggerate or misunderstand what their philosophical anthropology and ontology of hylomorphism has to offer here. Aristotelianism is not in the business of conducting experiments or correcting clinical diagnoses. Its role is more sapiential; it offers a big picture perspective of the human person as whole situated within a social context and exercising various psychological powers, which are hylomorphically constituted from various biological systems. Let us set forth some of these sapiential distinctions that can be operationalized in our philosophical, ethical, and scientific enquiries concerning dementia.

First is the distinction between personhood and personality. Personhood concerns the ontological identity of a developing dependent rational animal from the beginning to the end of its natural life. The rational animal’s personhood grounds the biological and psychological powers that enable the conscious life and bio-psycho-social-cultural developmental identity of the rational animal’s personality, including the developments of their collaborative theoretical, practical,
existential enquiries and stances. Independent of any acute or chronic illnesses, injuries, or diseases, a human person’s personality changes dramatically throughout their life, yet they remain one and the same human person whose dignity qua human person remains inviolable. Justice pertains as much to a developing human person no matter their realized and unrealized potentialities as it does to the developing human person with some unrealizable potentialities because they are or have become permanently disabled. None of these changes in personality undermine or alter the identity of the personhood of the developing rational animal. This is the person to whom justice requires that we must treat fairly and with dignity in accordance with the common good. Aristotelians contend that we cannot articulate an adequate ethical theoretical enquiry without first grasping the non-theoretical nature of the lived practices of human beings as developing dependent and often acutely or chronically disabled rational animals. And second, integrating these non-theoretical insights into a theoretically unified vision of the interdependency of ontological, biological, psychological, social, and so ethical factors.\textsuperscript{xiii}

Next, and to this enormously ambitious end, we need to distinguish between \textit{psychological level} powers and activities from the \textit{subpsychological level} biological systems that hylomorphically constitute all embodied psychological powers and operations. The relation between these two integrated phenomena are to be understood \textit{hylomorphically}; the distinction between psychological and subpsychological phenomena \textit{is not} to be construed as a terminological substitute for the mental-physical distinction—as it is for \textit{physicalists} (e.g., identity-reductionists or functionalist and anomalous monist non-reductionists) and \textit{dualists} (e.g., epiphenomenalists, emergentists, panpsychists, and interactive substance dualists). What does this contention mean? Briefly: tracking the distinction between personhood and personality, Thomists and other Aristotelians distinguish between two kinds of hylomorphic composition. First, there is the most fundamental hylomorphic composition of a substance. Second, all hylomorphic substances ground a panoply of embodied attributes, which are also hylomorphically constituted. So, in the case of a human, its substance is constituted by a rational soul that is the substantial form of the material body of a rational animal. The hylomorphic substance and personhood of the rational animal grounds its embodied and hylomorphically constituted psychosomatic powers and operations. But it also grounds, according to Thomists, its disembodied noetic powers and operations. The embodied psychosomatic powers and operations are \textit{psychological phenomena} that are hylomorphically constituted from \textit{subpsychological} biological systems, like the brain. For example, neural systems in the brain are formally organized material attributes of the animal which hylomorphically constitute the psychosomatic powers and operations of the animal. Very crudely: neural systems in the medial temporal lobe hylomorphically constitute the psychosomatic powers of memory. Neural systems comprised of the retina, optic nerve, lateral geniculate nucleus, occipital lobe and so-called visual ventral and dorsal streams are integral to the psychosomatic power of visual perception. But these complex \textit{subpsychological} biological systems—like the central and peripheral nervous systems, endocrine, muscular and skeletal systems, etc.—are themselves hylomorphically constituted from various levels of biological sub-systems and ultimately bio-chemical, chemical, and physical sub-systems. These psychological and sub-psychological systems nested within sub-systems are comprised of complex networks of coordinated manifestations of the powers of higher level systems actualizing, ordering, and directing the coordinated manifestations of its components or lower level systems, and vice-versa.

Third, and returning to the issue of intentional action, a Thomist can say that the embodied intentional actions of rational animals (e.g., running) are hylomorphically unified from the:

- Intellect and will \textit{qua} practical reasoning and willing which formally actualize, coordinate, \& direct
- the coordinated manifestation of the relevant psychosomatic powers of sensation, perception, motivation, executive registrations, and motricity required for the \textit{psychological dimension} of any embodied intentional action.
- This coordinated manifestation of psychosomatic operations are hylomorphically constituted from \textit{sub-psychological} biological systems—like the nervous, endocrine, cardiovascular, respiratory, muscular, and skeletal systems—which are themselves constituted from the coordinated manifestation of the myriad lower-level powers of these sub-psychological biological systems.
• Whenever the noetic operations actualize, order, and direct the coordinated manifestation of some psychosomatic powers’ operations, the coordinated manifestation of these psychosomatic powers’ operations thereby enlists the coordinated manifestation of the powers of the sub-psychological biological systems (and sub-systems, and sub-sub-systems) that hylomorphically constitute these psychosomatic powers and operations.

In order to fill out the details of these biological systems that hylomorphically constitute these psychosomatic powers and operations we can find some extraordinary resources in the empirically rich studies of the new mechanist philosophers (NMP) of biology, neuroscience, and psychology. Elsewhere I have argued for the compatibility of hylomorphism with the new mechanists philosophy of the special sciences; I take that for granted here. Hylomorphism supplies the ontology, and the philosophies of science (especially of biology, neuroscience, and psychology) provide the theoretical and hermeneutical mediation needed to appreciate the discoveries and rival interpretations and research programmes within scientific enquiries.

The new mechanist philosophy (NMP) of science focuses on four features that characterize biological mechanisms as they understand them: (1) component entities, (2) their activities, (3) organization, and (4) phenomena. (See figure #) “Mechanisms are entities and activities organized such that they exhibit the explanandum phenomenon.” The organized components of these biological mechanisms are themselves constituted from the organization of components of sub-level mechanisms, and so on and so forth. The functional operation of the higher level biological mechanisms depends on the functioning of the sub-mechanisms that its organized components are constituted from.

The ubiquity of mechanism dependence is a consequence of the hierarchical organization of mechanical systems and processes, and it explains how productive continuity at lower levels in the mechanistic hierarchy give rise to higher level forms of production. Productive powers of wholes derive from the organization of their parts, and the productive continuity of causal processes derives from productive interactions of parts at various stages in the process. (Glennan 2016, 811)

The organized component entities and their activities of a higher-level mechanism can only produce or underlie or maintain a phenomenon so long as these organized component entities and activities can enlist the powers of the lower-level mechanisms that each of these organized component entities and activities are composed from.

The behavior of the whole is dependent on the behavior of the components in such a way that interventions to change the components can change the behavior of the whole and vice versa. …One can disrupt spatial memory by ablating the hippocampus or knocking out NMDA receptors. (Craver, Explaining the Brain, 2007, 183)

Given my earlier Thomistic hylomorphic sketch of the way psychosomatic powers are constituted from the formal organization of biological systems (like the neural and glial systems of the brain), I hope a relatively straightforward image of the relevance of the new mechanist philosophy of neuroscience to Thomist hylomorphism is starting to come into view. Central to this hylomorphic framework—in contrast to emergentism—is the thesis that the sub-psychological level biological systems, like those in the brain, hylomorphically constitute, condition, and enable the psychological level psychosomatic powers and operations of animals.

Just as Aquinas recognized that lesions in the brain could cause impairments to phantasia that adversely affect the human person’s ability for intellectual understanding, so too we can draw upon the latest in work in neuropsychology to make more precise explanations—or at least complementary explanations at a variety of different levels—for various psychological deficits. Consider the following very conjectural sketch for how the psychological and sub-psychological effects of Alzheimer’s disease fit within a Thomist-Aristotelian anthropology. Pathological neural cell death is believed to result from the intracellular and extracellular accumulation of beta-amyloid plaques and neurofibrillary tangles of the tau protein (among others). Not only can these buildups disrupt intracellular processes they can also adversely affect microglia and lead to the sustained inflammation of the neurons which can then be targeted for neuronal cell death by the immune
system.\textsuperscript{xxix} All of these progressive neuronal and glial pathologies result in significant atrophy of neuro-glia systems. Consequently, the degeneration of various organized components of neurons leads to the degeneration of the neural systems constituted from these neurons. (Of course, this is extraordinarily simplified as I am leaving out a host of genetic and other intracellular and extracellular mechanisms and the entire scaffolding of multilevel mechanisms that bring us from neurons to neural systems.) So, for example, the atrophy of the medial temporal lobe (including the hippocampus) from such deteriorating neural systems impedes the optimal functioning of the personal or psychological level psychosomatic powers and operations. This is because these psychosomatic powers are themselves constituted from and enabled by the organization of neural networks distributed throughout areas of the brain like the medial temporal lobe. Psychosomatic powers like memory will be seriously hindered insofar as the manifestation of psychosomatic operations of recall, reminiscence, and autobiographical narration depend upon enlisting the organized causal components of these now atrophied sub-psychological neural systems. When the rational animal’s psychosomatic capacities for recollection, reminiscence, and autobiographical narration are inhibited by the atrophy of its sub-psychological neural systems, then not only will psychosomatic acts of recollection be obstructed, so also will all forms of reminiscence and autobiographical narration that are otherwise governed and directed through interactions with intellectual operations. Intelligent narration that relies upon psychosomatic capacities for reminiscence will be thwarted whenever reminiscence is impeded. So, if reminiscence cannot be exercised—because it cannot enlist the capacities of the crucial neural systems that constitute memory due to the neurodegeneration of the medial temporal lobe—then acts of reminiscence cannot be enlisted and guided by noetic operations, and intelligent narration becomes disabled.\textsuperscript{xxix}

In short, even though the deleterious effects of neurodegenerative disorders directly impact the psychosomatic powers and operations constituted from these deteriorating neural systems, they only indirectly impede intellectual powers and operations by inhibiting their capacity to formally actualize, govern, order, and direct psychosomatic operations. Any human activities that require the integrated confluence of noetic and psychosomatic operations—which for Thomist anthropology includes all intelligent activities of theoretical and practical reasoning and intentional action in this life—will be obstructed by damage to the sub-psychological neural systems that constitute and enable the psychosomatic powers of the rational animal.

4. Concluding Remarks. Psychosocial and Ethical Aspects of Dementia Care

I want to conclude by briefly, and contentiously, remarking on some of the practical and social implications of these seemingly quixotic Aristotelian speculations. We have seen that the psychological activities that comprise our \textit{personality} can be hindered, impeded, transformed, or undermined by the radical degeneration of the subpsychological biological systems that hylomorphically constitute our psychological activities. But we have also seen that our psychological activities—both intentional and non-intentional ones—involves the actualization, ordering, and directing of the coordinated manifestation of the powers of our subpsychological biological systems. The host of psychologically driven epigenetic factors that are activated whenever we engage in certain kinds of intentional and non-intentional behaviors are significant and remain an area of largely unexplored territory.\textsuperscript{xxxi} These metaphysical points provide us with practical orientations. How so?

When a patient is diagnosed with Alzheimer’s disease, or frontotemporal dementia, or any other disease, it provides family, friends, and even oneself with some explanation that makes sense of what were otherwise unintelligible actions. It also gives them some vague outlines of what to expect in the future and some directions for the patient and family to cope with the disease and its progression. Even if painful, knowing these truths can be a good. However, there are also certain psychological and social problems that lurk within any diagnosis. Ian Hacking has done much to illustrate for us a phenomenon he calls the “looping effect.”\textsuperscript{xxxii} Roughly when someone or a group is classified in a certain way, they often begin to act according to however they understand this
classification. This can thereby have the looping effect of making them fit the norms of a classification more than they really do or need to. So if the individual, family, or caregivers can only understand the diagnosis of dementia as a stigmatized classification, with fixed norms of progressive cognitive, conative, social, and personal disabilities, then the individual, family, and caregivers might begin to intentionally act according to these impoverished psychosocial norms. In other words, a stigmatized diagnostic classification can encourage psychosocial behaviors that implicitly re-enforce certain disabling expectations and norms of behavior. This can manifest itself with the individual or the family just giving up, and this psychosocial apathy can hasten the progression of the biopsychosocial factors of the disease. But it can also manifest itself in lack of patience with the individual’s growing inabilities or even misplaced sincere ways of trying to help—like making a meal, the bed, or putting on clothes. These are little things, but they might be the only things a patient can still purposefully perform on their own, and we should not take them away from them. If caregivers presume the individual is incapable of performing certain routines or practices that they actually can perform—even if less efficiently or with difficulty—they also can unintentionally hasten the growing psychosocial disabilities of the individual. To resist these often quite innocent and unintentional ways of reinforcing these deleterious looping effects, we need to transform how we interpret the psychosocial norms of these diagnostic classifications. That is to say, we need to think seriously and honestly about what the cardinal virtues demand of us either as individuals diagnosed with dementia or as family members, friends, or caregivers of those diagnosed with dementia. Throughout our lives—but especially when we are children, ill, or aging—we depend upon others to act as rational proxies, both to make decisions for us when we are not able to and to provide kinds of enabling co-operative practical reasoning which we participate in but are incapable of exercising on our own. Others are patiently enabling us to exercise our powers for intentional action by helping us to participating in their rationally ordered intentional actions. If our caregiving focuses on these kinds of co-operative enabling activities, then we enable the person to engage with us in purposeful intentional actions, which actualize, order, and direct the coordinated manifestation of their own sub-psychological biological systems. Again, the kinds of epigenetic factors actualized in these process are not immaterial; practiced resilience over apathy and indifference can have enormous effects on the progression of a disease.xxxxiii Consequently, what is required is more self-reflective caregiving and narratives of care that focuses on “person-centered care” and psychosocial engagements that aim to enable reliance and as much active participation as the patient is able and willing to exert in different contexts and at different times. But caregivers will only be able to achieve this insofar as they set for themselves virtuous exemplars and narratives of care ordered towards shared common goods. Justice is required to give what is due to every human person no matter their disabilities. Temperance requires that we do not unduely prioritize our own self-interests or those of other groups to the detriment and neglect of the disabled who often cannot voice their own needs and desires. We need courage to have resilience in the face of frustrations, failures, and the grief of seeing our loved one lose their cognitive and conative abilities. Finally, we need prudence to rightly direct our practical reasoning in these difficult times to find the golden mean between doing too much or not enough to help enable those we love suffering from dementia to still participate, as much as they can, in co-operative intentional activities.
Appendix

Dementia in General

Let us take a step back and consider dementia in general. Dementia describes a syndrome, that is, a collection of symptoms and signs; the etiology or causes of the disease are many and their connection to the syndrome complex. Significantly, it is an acquired disorder. Even though many cases of dementia have a genetic etiological profile, it is not a congenital disease; the disease is manifested late in life—though the quite rare early onset cases of dementia can occur quite early to individuals in their 30s—and so results in the loss of abilities, competences, and skills that the person once possessed. Dementia is also progressive, that is, the deterioration of one’s nervous system and psychological capacities gets worse and worse and seldom plateaus. Dementia has deleterious effects on both our psychological competencies and the sub-psychological biological systems that hylomorphically constitute, condition, and enable these psychological capacities and operations.

Aside from the damage done to our nervous system, which eventually can adversely affect our interconnected endocrine and immune systems, dementia can also give rise to such lived-body (Leib) complications as epileptic fits, ataxia and or deficits in motor skills, from jerkiness (myoclonus), gait abnormality and other abnormal movements up to immobility, insomnia, incontinence, swallowing problems (dysphagia), weight loss, and so forth. And, more stereotypically, the deterioration to the brain caused by dementia takes its toll on our embodied cognitive and conative capacities and thereby modify our personality. Characteristically, this includes such deficits in higher cognitive capacities such as amnesia in semantic or episodic memory, Wernicke’s aphasia and Broca’s aphasia (that is, deficient in linguistic comprehension and production (dysphasia), respectively), recognition, an inability to concentrate or pay attention, difficulties with planning (working memory, fluid intelligence, executive function), problems with calculating, abstract thinking, and so forth. Significantly, with the exception of Lewy Body Dementia, most forms of dementia are not directly connected to symptoms of clouded consciousness or fuzzy awareness. Finally, dementia is also generative of a constellation of conative, motivational, and temperamental phenomena, such as aggression, anxiety, agitation, irritability, disinhibition, apathy, depression, dysphoria, eating disorders, shouting, delusions, hallucinations, and “wandering.” As Julian Hughes points out, although dementia is a “brain disorder, [it] is one that profoundly affects the whole body and the whole person.” The adverse effects dementia has on our psychological and sub-psychological abilities, thereby altering our personality, spiral into other maladies influence the existential, social, and political dimensions of the human person. As the disease progresses the person with dementia becomes completely dependent on others, and the cost to families, society, and the government are tremendous. Beginning with the last two, the world costs of dementia related healthcare in 2010 were estimated to be US$604 billion, which is about 1 percent of the world’s gross domestic product. There estimated to be about 36 million people in the world with dementia in 2010, there is a projected 65 million by 2030 and 115 million by 2050, a 225 percent increase in the 40 years between 2010 and 2050. But more importantly, especially for our concerns, is dementia’s heavy hit on the psychological and subpsychological capacities that comprise the personality and lived experiences of the human person. As the disease progresses it slowly isolates the human person and estranges them from knowing and appreciating their family, friends, the meaning of what they have done, what they have lived for, and who they are in the full existential significance and gravitas of these characterizations. Dementia leads to the breakup of the coherent self-interpreting narrative that is partially constitutive of our lives as human persons. As Hughes sagaciously observes, “There is no doubt that dementia, like many other diseases, is cruel. Society should not make that worse. If we focus on the whole picture—the whole person in his or her social setting—we are more likely to get things right.”

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1 These run the gamut of rather extreme pluralists, to new mechanist philosophers, bio-psycho-social models, to Dominic Murphy’s sympathies with integrative pluralism. See Mitchell, S. ‘Taming Causal Complexity.’ Philosophical
I am extraordinarily grateful to two philosopher clinicians, Dr Julian Hughes, an old age psychiatrist, and Dr Christopher Butler, a cognitive neurologist, who have both generously shared so much of their research and clinical experiences with me over the past four years. Any deficiencies in my portrayal of the medical research or clinical practice concerning dementia are entirely my own.

“Explanation in Psychiatry” 604.

I have set aside here the tricky and contentious issues of making perspicuous the needed stipulated theoretical distinctions between voluntary, involuntary, and nonvoluntary actions and their nexus with intentional, unintentional, and nonintentional. Roughly, what we do (or allow to happen or suffer) intentionally is done on the basis of reasons for acting, the reasons with which one important consequence. There is no such thing as ‘behavior’, to be identified prior to and independently of intentions, beliefs and settings. Hence the project of a science of behavior takes on a mysterious and somewhat outré character. It is not tantamount to a constraint on acceptable explanation imposed on inquiry in advance. It is just as unwarranted to suppose that the relevant explanatory variables for psychotic or affective phenomena must be cognitive as it is to adopt a simple reductionism and just argue that they must be biological. The fact is that decades of research have revealed that mental illness is caused by, and effects in turn, ever so many different physical, psychological, and social factors. We simply have to take these causal relations where we find them. There seems no need to complicate an already horrible epistemic task by insisting on some constraints on explanation, arguing that causal relations must really be something or other, in the name of some prior metaphysical commitment or theory of psychological explanation.” Murphy, “Explanation in Psychiatry” 604.
Hughes, following Sabat draws attention to the sometimes malignant ways in which these tests can be—but need not be—administered, ways that often unhelpfully categorize (i.e., position) and stigmatize the patients that perform them. “Positioning is evident even in the benign-seeming procedure of cognitive testing. As Sabat points out, psychometric testing which places the person below a statistically derived cut-off immediately positions the person with acquired diffuse neurocognitive dysfunction as inferior in some sense (Sabat, 2001: 166). Cognitive tests amount, therefore, to ‘defectology’: they show what the person cannot do, not what they can. Indeed, we know that formal cognitive testing can be upsetting both for the person afflicted with the condition and for the informal carer: having admitted that she and her husband had cried as a result of the testing, one spouse told me in an interview that she wondered why her husband could not have been more simply engaged in a conversation about politics to test his mental abilities (Hughes, Hope, Reader, and Rice, 2002). We also know that stigma can immediately follow diagnosis, which can also be described as harrowing.” Hughes, Thinking Through Dementia, 232.

Dominic Murphy has argued that the “variety in mental illness requires us to explain psychiatric phenomena not by looking for stable regularities but by constructing exemplars. … [A]n exemplar [is] an imaginary patient who has the ideal textbook form … of a disorder, and only that disorder. The idea is to model all the causes that contribute to a natural history so as to explore how they work together in different contexts to produce the various outcomes of interest, and then understand patients as idiosyncratic instances of the exemplar. The exemplar lets us identify robust processes (Sterely 2003:131–2, 207–8) that are repeatable or systematic in various ways, rather than the actual processes that occur as a disorder unfolds in one person. But we do not stop there: The ultimate goal is causal understanding of a disease. We build a model to serve this end. It aims to represent the pathogenetic process that accounts for the observed phenomena in the exemplar. To explain an actual history in a patient is to show how the processes unfolding in the patient resemble those that are assumed to occur in the exemplar. Exemplars provide an idealized form of the disorder that aims to identify the factors that remain constant despite all the individual variation.” Murphy, “Explanation in Psychiatry” 608-609.

The three clinical variants of frontotemporal dementia are the frontal (behavioural) variant, semantic dementia, and progressive non-fluent aphasia. “Pick bodies and cells are characteristic pathological findings in many cases and involve tau; but tauopathies (Pick bodies and neurofibrillary tangles) only account for 45 percent of the pathology; most of the rest is accounted for by transactive response DNA binding protein (TDP-43). Mutations on the genes on chromosome 17 which code for tau and progranulin account for 20 percent of frontotemporal lobar degeneration.” Hughes, Alzheimer’s and other Dementia, 61-66.

“In FTD (the frontal, behavioural variety) there is bilateral atrophy (i.e. shrinkage) of the frontal and anterior temporal lobes (i.e. the front parts of the temporal lobes); In semantic dementia there is bitemporal atrophy (i.e. shrinkage affecting both sides), but the atrophy is often worse on the left and tends to be anterior rather than posterior; In progressive non-fluent aphasia there is marked asymmetry, most often affecting the left hemisphere around the area that serves speech production…. Although approximately 45 percent of cases of FTLD show evidence of tau in the brain, either as [neurofibrillary tangles] NFTs or as Pick bodies, the further 55 percent (approximately) of cases can be accounted for by a type of pathology known as FTLD-U, because instead of tau it contains a protein that stains for ubiquitin. This protein is called the transactive response DNA binding protein, abbreviated to TDP-43. The details of the TDP-43 protein do not need to concern us here. Together, the pathology caused by tau (taupathies) and the pathology linked to TDP-43 account for 95 percent of all cases of FTLD. A very small proportion of cases is accounted for by other proteins.” Ibid., 65. “The observation that in many families (perhaps 40 percent) these conditions appear to have an autosomal dominant pattern of inheritance has led to a search for the responsible gene or genes. The most significant finding to date relate to chromosome 17. This is the chromosome that codes for tau. And, indeed, mutations (i.e. changes in the genetic code) have been found in the tau gene, which is known as the MAPT gene (which stands for microtubule associated protein tau). Further mutations were then found in the progranulin gene (PGRN). Progranulin is connected with tissue remodelling or repair, and seems to be important for neuronal survival. The PGRN gene is very close to the MAPT gene. But together the MAPT and PGRN mutations only account for about 20 percent of all cases of FTLD. Work continues in this field.” Ibid. 65-66. Hughes presents a more focused overview of these pathologies affecting different forms of dementia, in chapter 4.

“Frontotemporal dementia (FTD) encompasses a spectrum of neurodegenerative diseases with heterogeneous clinical presentations and two predominant types of underlying neuropathology. FTD typically comprises three distinct clinical syndromes: behavioral variant frontotemporal dementia (bvFTD), semantic variant primary progressive aphasia (svPPA), and nonfluent variant primary progressive aphasia (nfvPPA). FTD also frequently overlaps both clinically and neuropathologically with three other neurodegenerative syndromes: corticobasal syndrome (CBS), progressive supranuclear palsy (PSP), and amyotrophic lateral sclerosis (ALS). Each syndrome can be associated with one or more underlying neuropathological diagnoses and are referred to as frontotemporal lobar degeneration (FTLD). Although the various FTD syndromes can substantially differ in terms of clinical symptoms and underlying pathology, the syndromes can be broadly categorized into behavioral, cognitive and motor domains. Currently there are no Food and Drug Administration (FDA) approved therapies for the above syndromes except riluzole for ALS. FTD treatment strategies generally rely on off-label use of medications for symptomatic management, and most therapies lack quality evidence from randomized, placebo-controlled clinical trials. For behavioral symptoms, selective serotonin reuptake inhibitors may be effective, while case reports hint at possible efficacy with antipsychotics or antiepileptics, but use of these latter agents is limited due to concerns regarding side effects. There are no effective therapies for cognitive complaints in
FTD, which frequently involve executive function, memory, and language. Motor difficulties associated with FTD may present with parkinsonian symptoms or motor neuron disease, for which riluzole is indicated as therapy. Compared to idiopathic Parkinson’s disease, FTD-related atypical parkinsonism is generally not responsive to dopamine replacement therapies, but a small percentage of patients may experience improvement with a trial of carbidopa-levodopa. Physical and occupational therapy remain an important corner stone of motor symptom management in FTD. Speech therapy may also help patients manage symptoms associated with aphasia, apraxia, and dysarthria. Recent advances in the understanding of FTLD pathophysiology and genetics have led to development of potentially disease-modifying therapies as well as symptomatic therapies aimed at ameliorating social and behavioral deficits.” Tsai, R. M., & Boxer, A. L. (2014). Treatment of frontotemporal dementia. *Current treatment options in neurology*, 16(11), 319.


xiii But for important limitations of NMP, especially when it comes to psychological phenomena and specifically psychiatric phenomena, see Dominic Murphy, “Explanation in Psychiatry,” p. 603: “It seems that something like this ought to work in psychiatry. Psychiatric phenomena are clearly, in way or another, things that go wrong with human animals, and if explanation in the life sciences involves mechanisms, we should expect mechanistic explanations, and given the variety of causal influences we find, we probably need to think about levels of explanation too. But the way these concepts are understood in other parts of the philosophy of the inexact sciences may not fit psychiatry straightforwardly. It is not just that psychiatrists need to look for something less like laws and more like mechanisms. It also seems to be the case that mental illnesses often involve many different kinds of cause that interact in ways that do not fit the idea of parts of mechanism entering into causal relations that are regularly productive of phenomena. And a widespread assumption in philosophy of psychology is that levels of explanation are different ways of picking out the same phenomenon – a computational process realized in brain tissue, for example. But in psychiatry, as we will see, in a moment, variables at different levels relate different phenomena, not the same phenomenon described in different ways.” See also, Murphy, ‘Levels of Explanation in Psychiatry.’ *Philosophical Issues in Psychiatry*. Eds Kenneth Kendler and Josef Parnas. Baltimore, MD: Johns Hopkins University Press, 2008. 99–125


xxv As I understand it, hylomorphism provides an ontological framework for understanding inanimate and animate things. Thomist philosophical anthropology deploys this hylomorphic ontology in its enquiries into the ethical, social, psychological, and biological factors that constitute human persons. However, when it comes to the biological details of the sub-psychological systems that materially constitute rational and other animals, these are investigated by the special sciences, not by philosophical anthropology. This is where philosophy of the special sciences come in; they provide a fruitful mediating role between more abstract ontological investigations and the highly detailed concrete experimental investigations of the special sciences. What the new mechanist philosophers provide Thomist hylomorphism then, are theoretical resources for thinking more concretely about how the discoveries of neuroscience, for instance, might fit or resist fitting within any one of the many rival ontological accounts of human beings.


xxvii For instance, impediments to the causal processes of certain mechanistic components critical for higher-level psychosomatic operations, say, dorsolateral frontal lesions that adversely affect executive registrations, will thereby indirectly hinder the human person’s ability to perform any intellectual operations that interact with, and so require the integrity of, such psychosomatic operations as executive registrations. Any unified activity of the human person requires the integrated operations of diverse reciprocal powers. Intellectual operations of practical reasoning and intentional action can only formally govern and direct executive registrations, memorative recollections, and the apprehension or articulation of intelligent speech if they are integrated with these psychosomatic operations. But there can be no such interaction between intellectual and psychosomatic operations if there are impediments to the exercise of these psychosomatic capacities. Impediments can arise at the psychological level of these psychosomatic capacities for perceptual registration, memory, and speech as well as at the sub-psychological level of the neural, genetic, and biochemical systems and subsystems that constitute and enable the proper functioning of these psychosomatic capacities.

xxviii See *ST* I.78.4; 84.7 89.5. See Craver 2007; Kendler, Zachar, and Craver 2011; Hughes 2011.

xxix “Initially described almost 100 years ago by Alois Alzheimer, [Alzheimer’s Disease] AD is one of the most common age-related neurodegenerative diseases, with approximately 7% of people older than 65 years and about 40% of people older than 80 years being affected in industrialized countries. The symptoms of AD are characterized by loss of memory, progressive impairment of cognition, and various behavioral and neuropsychiatric disturbances. The pathological hallmarks of AD in the brain include extracellular amyloid plaques comprising aggregated, cleaved products of the amyloid precursor protein (APP) and intracellular neurofibrillary tangles (NFTs) generated by
hyperphosphorylated forms of the microtubule-binding protein tau. Evidence of an inflammatory response in AD includes changes in microglia morphology— from ramified (resting) to amoeboid (active)—and astrogliosis (manifested by an increase in the number, size, and motility of astrocytes) surrounding the senile plaques. Moreover, microglia surrounding plaques stain positive for activation markers and proinflammatory mediators, including MHC class II, Cox-2, MCP-1, TNF-α, IL-1β, and IL-6 (Akiyama et al., 2000). MCP-1 is known to induce the chemotaxis of astrocytes and contributes to the recruitment of astrocytes around senile plaques (Wyss-Coray et al., 2003). In addition, elevated levels of chemokines and cytokines and their receptors, including IL-1α, CXCR2, CCR3, CCR5, and TGF-β, have been reported in post-mortem AD brains (Cartier et al., 2005).” P. 4.

“Sustained inflammation resulting in tissue pathology implies persistence of an inflammatory stimulus or a failure in normal resolution mechanisms. A persistent stimulus may result from environmental factors or the formation of endogenous factors (e.g., protein aggregates) that are perceived by the immune system as “stranger” or “danger” signals. Inflammatory responses that establish feed-forward loops may overwhelm normal resolution mechanisms. Although some inflammatory stimuli induce beneficial effects (e.g., phagocytosis of debris and apoptotic cells), and inflammation is linked to tissue repair processes, uncontrolled inflammation may result in production of neurotoxic factors that amplify underlying disease states.” Glass CK, Saijo K, Winner B, Marchetto MC, Gage FH. Mechanisms underlying inflammation in neurodegeneration. Cell. 2010;140(6):918-34.


xxxii Steven Sabat Alzheimer’s Disease and Dementia: What Everyone Needs to Know (OUP, 2018), chapter 5, “Resilience, Selfhood, and Creativity”

xxxv Cf. Julian Hughes, Alzheimer’s and other dementias, Julian Hughes, Thinking Through Dementia (OUP, 2011); Julian Hughes, How we think about dementia: personhood, rights, ethics, the arts and what they mean for care (Jessica Kingsley Publishers, 2014).

xxxvii Hughes, Alzheimer’s and other dementias, 27.

xxxviii Hughes, Alzheimer’s and other dementias, 11.