

its disappearance the interest of social science, policy-makers, and the different groups that make up this strata to develop a broad definition based upon the distinction between 'us,' the 'white-collar,' and 'them,' the 'blue-collar workers,' seems to have vanished. What remains is a remarkable gap between the numerical importance of the group in question and its relative neglect by social scientists in the last decades.

See also: Bourgeoisie/Middle Classes, History of; Income Distribution; Professionalization of Social and Behavioral Scientists: United States; Professionalization/Professions in History; Professions, Sociology of; Social Inequality in History (Stratification and Classes); Social Mobility, History of; Work and Labor: History of the Concept; Work, History of; Working Classes, History of

Bibliography

- Anderson G I 1976 *Victorian Clerks*. University Press, Manchester
- Bain G S 1972 *The Growth of White-collar Unionism*, 2nd edn. Clarendon Press, London
- Bain G S, Pollins I I 1965 The history of white-collar unions and industrial relations: A bibliography. *Bulletin of the Society for the Study of Labour History* 11: 20–25
- Behringer P 1985 *Soziologie und Sozialgeschichte der Privatangestellten in Großbritannien*. Frankfurt, Germany
- Bonham J 1954 *The Middle Class Vote*. Faber of Faber, London
- Coyner S J 1977 Class consciousness and consumption: The new middle class during the Weimar Republik. *Journal of Social History* 10: 310–31
- Croner F 1951 *Tjänstemannakaren i det moderna Samhället*. Stockholm (German, revised edn. Frankfurt 1954)
- Crossick G (ed.) 1977 *The Lower Middle Class in Britain 1870–1918*. St. Martin's Press, New York
- Crozier M 1971 *The World of the Office Worker* (trans. Landau D). University of Chicago Press, Chicago
- Falter J W 1991 *Hitlers Wähler*. Beck, Munich
- Fourastié J 1963 *Le grand espoir du XXe siècle*. Gallimard, Paris
- Fromm E 1984 *The Working Class in Weimar Germany—a Psychological and Sociological Study* (trans. Weinberger B, ed. and with an introduction by gang Bonss W). Bierg, Leamington Spa, UK
- Gerstl J E, Ilutton S P 1966 *The Engineers: The Anatomy of a Profession. A Study of Mechanical Engineers in Britain*. Tavistock Publications, London
- Giddens A 1981 *The Class Structure of the Advanced societies*, 2nd edn. Hutchison, London
- Hilferding R 1981 *Finance Capital: A Study of the Latest Phase of Capitalist Development* (ed. with an introduction by Bottomore T from translations by Watnick M, Gordon S). Routledge, London
- Humphreys B V 1958 *Clerical Unions in the Civil Service*. Blackwell & Mott, London
- Jenkins C, Sherman B 1979 *White-collar Unionism. The Rebellious Salariat*. Routledge, London
- Kater M H 1983 *The Nazi Party—a Social Profile of Members and Leaders, 1919–1945*. Blackwell, Oxford, UK
- Kocka J 1980 *White-collar Workers in America: 1890–1940: a Social-political History in International Perspective*. Sage, London
- Kocka J (ed.) 1981 *Angestellte im europäischen Vergleich. Die Herausbildung angestellter Mittelschichten seit dem späten 19. Jahrhundert*
- Koenig M 1991 *Die Angestellten unterwegs. Vom Berufsstand zur modernen Gewerkschaft 1890–1990*. Bund-Verlag, Kuhn
- Kracauer S 1998 *The Salaried Masses—Duty and Distraction in Weimar Germany* (trans. Hoare Q and with an introduction by Mulder-Bach I). Verso, New York
- Lederer F 1979 *Kapitalismus, Klassenstruktur und Probleme der Demokratie in Deutschland 1910–1940. Ausgewählte Aufsätze von Emil Lederer herausgegeben von Jurgen Kocka*. Vandenhoeck und Ruprecht, Goettingen, Germany
- Lipset S M 1960 *Political Man: The Social Bases of Politics*, 1st edn. Doubleday, Garden City, NY
- Lockwood D 1989 *The Blackcoated Worker—a Study in Class Consciousness*, 2nd edn. University Press, Oxford, UK
- Mallet S 1963 *La nouvelle classe ouvrière*. Paris (Spanish: *La nueva condición obrera*. Madrid 1969).
- Mills C W 1951 *White-collar—the American Middle Classes*. Oxford University Press, New York, London
- Reader W J 1966 *Professional Men: The Rise of the professional Classes in 19th Century England*. Weidenfeld & Nicolson, London
- Schulz G 2000 *Die Angestellten seit dem 19. Jahrhundert*. Oldenbourg, Munchen, Germany
- Sobel R 1989 *The White-collar Working Class. From Structure to Politics*. Proeger, New York
- Speier II 1986 *German White-collar Workers and the Rise of Hitler*. Yale University Press, New Haven and London
- Suhr S 1930 *Die weiblichen Angestellten*. Zentralverband der Angestellten, Berlin
- Touraine A 1969 *La société post-industrielle*. Paris

M. Prinz

Williams Syndrome

In the final decades of the twentieth century, cognitive neuroscience paid particular attention to a group of disorders known as microdeletion syndromes. These are neurodevelopmental disorders in which the missing genes on a chromosome can be identified and the resulting gene-behavior relations explored. They differ from syndromes like Down syndrome in which an entire extra chromosome is present, as in Trisomy 21. One microdeletion disorder that caused particular excitement among cognitive neuroscientists is Williams syndrome (henceforth WS). This is because WS results in an unusually uneven cognitive profile. Language and face processing are seemingly spared, whereas other higher cognitive functions (spatial cognition, number, planning, and problem solving) are seriously impaired. Initial characterizations of the syndrome at the cognitive level seemed to hold the promise of relatively straightforward gene-cognition

mappings. WS was, and continues to be, hailed as the prime example of some intact, innately specified cognitive modules in the face of general intellectual impairment (e.g., Pinker 1994, 1999). However, more recent evidence fails to support this view, favoring instead a more dynamic, neuroconstructivist approach to genetic disorders. In this entry, the cognitive processes underlying the purportedly spared domains will be examined, together with a focus on the infant starting state and the developmental trajectory leading to the phenotypic outcome.

1. Williams Syndrome: Genetic Profile

Williams syndrome is a rare developmental disorder and occurs in approximately 1 in 20,000 live births. It is caused by a submicroscopic deletion on chromosome 7q11.23. The deleted region contains some 20 genes, about 17 of which have been identified (Ewart et al. 1993, Frangiskakis et al. 1996, Meng et al. 1998, Tassabehji et al. 1996). Only a few of these genes are expressed in the brain and are therefore of special interest to the cognitive neuroscientist. Others affect the physical development of patients, particularly with respect to impairments to the cardio-vascular system.

Initial excitement came from the discovery that in the majority of cases of WS one copy of the elastin gene (ELN) (Ewart et al. 1993) and one copy of the Limkinase1 gene (LIMK1) (Frangiskakis et al. 1996, Tassabehji et al. 1996) were consistently deleted. ELN is important for elasticity of the heart, skin, blood vessels, and lungs. Its deletion was therefore rapidly linked to the facial dysmorphology and supra-valvular aortic stenosis (SVAS) found consistently in individuals with WS (Ewart et al. 1993). LIMK1 is expressed in the developing brain and its deletion was claimed to explain the typical pattern of spatial impairments found in the cognitive profile of such individuals (Frangiskakis et al. 1996, Mervis et al. 1999). Despite the claims about the role in WS of the ELN and LIMK1 genes in facial dysmorphology and spatial cognition respectively, direct one-to-one genotype/phenotype mappings are highly unlikely in cognitive neuroscience, as a subsequent study by Tassebehji and collaborators showed (Tassabehji et al. 1996). Three patients with SVAS were examined who had partial deletions on chromosome 7 in the same region as the clinical groups with WS. The study showed that despite the ELN and LIMK1 deletions, none of these patients had the facial dysmorphology typical of WS, nor did they display the uneven WS cognitive profile of impaired visuo-spatial cognition and enhanced linguistic capacities. They all had an even cognitive profile within the normal range. The results indicate that the ELN deletion does not alone explain the facial dysmorphology found in WS. They also suggest that the LIMK1 deletion is either irrelevant to the development of spatial cognition or that its expression

interacts with a number of other genes to contribute to the spatial impairment. But it is clear that one-to-one mapping between specific genes and higher cognitive outcomes does not hold.

2. Williams Syndrome: Phenotypic Profile

The pioneering work of Bellugi and her collaborators initially pointed to some clear-cut dissociations in the cognitive architecture of WS. Language and face processing appeared to be surprisingly preserved in the face of both general retardation and particularly serious problems with visuo-spatial cognition, number, planning, and problem solving (Bellugi et al. 1994). Researchers in the field of WS have been fairly cautious about their claims, referring to relative strengths and weaknesses rather than absolute ones (Bellugi et al. 1999, Karmiloff-Smith 1998, Klein & Mervis 1999, Mervis 1999, Tager-Flusberg et al. 1998, Vicari et al. 1996, Volterra et al. 1996). By contrast, secondary sources cited in writings by linguists, developmental psychologists, neuropsychologists of adult brain damage, and philosophers have often used Williams syndrome to bolster claims about innate and independently functioning modules, some of which are purportedly intact and others impaired (e.g., Pinker 1994, 1999). This stems from the view that the pattern of behavioral performance found in the phenotypic outcome is a direct window on the purported innately specified, modular structure of the cognitive architecture of the brain (Baron-Cohen 1998, Leslie 1992, Temple 1997). Such reasoning treats the genetically impaired brain as if it were a normal brain with parts intact and parts impaired, ignoring the dynamic role of genetic mutation in interaction with environmental input in fostering overall brain growth. This has been particularly the case with studies of autism and Williams syndrome, in which cognitive impairments in older children and adults have been used to make claims about gene expression, in the absence of studies of the starting state in infants.

Recent studies of infants, children, and adults with WS strongly suggest that the starting state cannot be simply inferred from the phenotypic outcome (Pater-son et al. 1999). Infants with WS were compared with chronological (CA) and mental-age (MA) matched infants with Down syndrome (DS), as well as MA and CA-matched typically developing controls. Despite the fact that WS adults perform significantly better than DS adults on vocabulary tasks, WS infants are as seriously impaired on vocabulary tasks as are DS infants. Moreover, despite WS adults having significantly worse problems with judging numerosities in adulthood than DS adults and control groups, WS infants perform normally, like the CA-controls, and significantly better than DS infants. In other words, the patterns obtaining in infancy turn out to be the opposite of the patterns in adulthood, pointing to the

importance of the dynamics of developmental trajectories over time, rather than a static view of the infant starting state and the phenotypic outcome.

Even in cases where the phenotypic outcome seems to display spared performance, in-depth analyses suggest that people with WS process inputs via different cognitive processes (Karmiloff-Smith 1998). In the domain of vocabulary, individuals with WS do not obey the same lexical constraints as normal controls when learning new words. In the domain of syntax, WS adults tend to display patterns typical of much younger children rather than intact performance (Klein and Mervis 1999). For example, it has been claimed that individuals with WS have intact regular past tense formation of verbs, alongside impaired associative lexical processes (Clahsen and Almazan 1998). Recent research challenges this claim in that once verbal mental age is taken into account, WS patients display no selective deficit for irregular verbs (Thomas et al. 2001). Specifically, the WS data can be placed on the normal developmental pathway found in much younger subjects. These various results are consistent with the hypothesis that the WS language system is seriously delayed because it has developed under different constraints.

WS language is not simply delayed, however. Several studies now suggest that there is an imbalance in WS at different times in development between phonology and semantics (Karmiloff-Smith 1998, Thomas et al. 2001). For example, when WS participants monitor sentences for a target word, they do not show sensitivity to certain sentential violations, suggesting that in WS semantic information may become available too slowly to be integrated with the online processing of syntax. A study of reading in WS came to similar conclusions about the weak role of semantics in learning to read new words. The WS group displayed equal levels of reading for both concrete and abstract words. By contrast, the controls found concrete, imageable words much easier to read. In general, imageability effects have been shown to be weaker in people with WS (Karmiloff-Smith 1998). A more recent study by Vicari and his colleagues demonstrated that, compared to normal controls, word learning is superior in WS if the auditory presentation of a word is accompanied by the simultaneous presentation of a photograph depicting the object (Vicari et al. 2000). This seems to be because, unlike normal controls, people with WS are defective in spontaneously forming a visual (semantic) image of auditorily presented words. Finally, when learning new spoken words and despite a vocabulary test age of 8, people with WS behave like 4–5 year olds, and do not show the pattern typical from 6 years onwards in the normal population. Like very young children, WS patients are less influenced by the semantics of the real words that the nonce terms resemble. Rather, they rely more on phonology. Taken together, a variety of studies suggest that, unlike typical development, phonological

representations are at times stronger than semantic representations in their influence on the way in which WS language develops.

Although it is becoming increasingly clear that vocabulary development does not follow a normal developmental trajectory in WS and that semantics places a weaker constraint on WS language development than in typical controls, it remains possible that WS syntax is intact, as many have claimed (e.g., Pinker 1994, 1999). There are, however, a number of lines of evidence to doubt this. First, vocabulary levels are usually better than syntactic levels in WS on various standardized tasks, although both are significantly below chronological age (Karmiloff-Smith 1998). Second, even in very simple imitation tasks, participants with WS show impairment with complex syntactic constructions such as embedded relative clauses. These and various other findings in different laboratories are hardly consistent with the view that WS syntax is intact. Even in an area of relatively simple syntax—grammatical concord over sentence elements—which young, normal French-speaking children acquire easily and early, people with WS show impairment even in adulthood (Karmiloff-Smith 1998). Although the WS children learn the local gender marker (correct article) for a nonce term easily (in fact, more easily than control children), their capacity for gender agreement across sentence elements such as agreement on adjectives or pronouns is seriously impaired. Studies of Italian-speaking children have also revealed that grammatical gender is a particular problem, with WS children displaying errors never encountered in normal development (Volterra et al. 1996). Several studies (e.g., Klein and Mervis 1999) now suggest that the problems that people with WS have with semantics and syntax are often camouflaged by their good verbal memory. This again demonstrates that overt behavior is not necessarily an index of underlying cognitive competence.

A similar conclusion holds for the domain of face processing. Despite reports that WS face processing is intact (Bellugi et al. 1994, Rossen et al. 1996), in-depth studies of face processing suggest that individuals with WS use different strategies from normal controls (Karmiloff-Smith 1998). Several studies (Deruelle et al. 1999, Karmiloff-Smith 1998, Udwin & Yule 1991) have replicated earlier work, revealing normal or near normal behavioral scores on standardized tasks like the Benton Facial Recognition Test (Benton et al. 1983) and the Rivermead Behavioural Memory Test (Wilson et al. 1985). However, these studies have seriously challenged the notion that the behavioral success displayed in WS face processing capacities is subserved by normal cognitive processes. Where normal controls use predominantly configural strategies for processing upright faces and featural strategies for processing inverted faces, the WS patients tend to process the featural details of both upright and inverted faces. These different strategies have been

further explored in brain imaging studies (Mills et al. 2000). When older children and adults with WS have to match faces in an event related potential study, they display temporal processes found at no age in normal controls. They also tend to process faces bilaterally or predominantly with the left hemisphere, whereas normal controls show a right hemisphere bias for faces. All of these findings point to atypical face processing in WS, despite the normal behavioral scores.

3. Concluding Comments

Developmental cognitive neuroscience must take development seriously. The WS brain is 20% smaller than normal brains and qualitatively different in terms of brain anatomy (Bellugi et al. 1999), brain chemistry (Rae et al. 1998), and computational processing (Mills et al. 2000). This holds throughout embryogenesis and postnatal brain development and means that interaction with environmental stimuli will be subtly different. Given a very different brain, it is unsurprising that even when overt behavior seems normal, as in some aspects of WS language and face processing, these skills actually turn out to be underpinned by cognitive processes that are different from the normal case (Karmiloff-Smith 1998).

Williams syndrome is an excellent model for the neurocognitive study of genetic disorders, because of its strikingly unusual cognitive profile. The syndrome is especially important because of the way in which in-depth research highlights the need to go beyond both observable behavior and static descriptions of snapshots of developmental outcomes, to the charting of neurocognitive trajectories from infancy onwards.

See also: Cognitive Development in Infancy: Neural Mechanisms; Infant Development: Physical and Social Cognition; Prefrontal Cortex Development and Development of Cognitive Function

Bibliography

- Baron-Cohen S 1998 Modularity in developmental cognitive neuropsychology: Evidence from autism and Gilles de la Tourette syndrome. In: Burack J A, Hodapp R M, Zigler E (eds.) *Handbook of Mental Retardation and Development*. Cambridge University Press, Cambridge, UK, p. 335
- Bellugi U, Wang P P, Jernigan T 1994 Williams Syndrome: An unusual neuropsychological profile. In: Broman S H, Grafman J (eds.) *Atypical Cognitive Deficit in Developmental Disorders: Implications for Brain Function*. Lawrence Erlbaum Associates Inc., Hillsdale, NJ, pp. 23–56
- Bellugi U, Lichtenberger L, Mills D, Galburda A, Korenberg J R 1999 Bridging cognition the brain and molecular genetics: evidence from Williams syndrome. *Trends in the Neurosciences* **22**(5): 197–207

- Benton A L, Hamsher K, de S Varney N R, Spreen O 1983 *Contributions to Neuropsychological Assessment*. Oxford University Press, New York
- Clahsen H, Almazan M 1998 Syntax and morphology in Williams syndrome. *Cognition* **68**(3): 167–98
- Deruelle C, Mancini J, Livet M O, Casse-Perrot C, de Schonen S 1999 Configural and local processing of faces in children with Williams syndrome. *Brain and Cognition* **41**: 276–98
- Ewart A K, Morris C A, Atkinson D, Weishan J, Sternes K, Spallone P, Stock D A, Leppert M, Keating M T 1993 Hemizyosity at the elastin locus in a developmental disorder Williams syndrome. *Nature Genetics* **5**: 11–6
- Frangiskakis J M, Ewart A, Morris C A, Mervis C B, Bertrand J, Robinson B F, Klein B P, Ensing G, Everett L A, Green E D, Proschel C, Gutowski N J, Noble M, Atkinson D L, Odelberg S J, Keating M T 1996 LIM-kinase1 hemizyosity implicated in impaired visuospatial constructive cognition. *Cell* **86**: 59–69
- Karmiloff-Smith A 1998 Development itself is the key to understanding developmental disorders. *Trends in Cognitive Sciences* **2**: 389–98
- Klein B P, Mervis C B 1999 Contrasting patterns of cognitive abilities of 9 and 10-years-olds with Williams syndrome or Down syndrome. *Developmental Neuropsychology* **16**(2): 177–96
- Leslie A M 1992 Pretence autism and the theory-of-mind-module. *Current Directions in Psychological Science* **1**: 18–21
- Meng X, Lu X, Zhizhong L, Green E D, Massa H, Trask B J, Morris C A, Keating M T 1998 Complete physical map of the common deletion region in Williams syndrome and identification and characterization of three novel genes. *Human Genetics* **103**: 590–9
- Mervis C B 1999 The Williams syndrome cognitive profile: Strengths weakness and interrelations among auditory short-term memory language and visuospatial constructive cognition. In: Winograd E, Fivush R, Hirst W (eds.) *Ecological Approaches to Cognition: Essays in Honor of Ulric Neisser*. Lawrence Erlbaum Associates Inc. Mahwah, NJ, pp. 193–227
- Mervis C B, Morris C A, Bertrand J, Robinson B F 1999 Williams syndrome: Findings from an integrated program of research. In: Tager-Flusberg H (ed.) *Neurodevelopmental Disorders*. MIT Press, Cambridge, MA, pp. 65–110
- Mills D L, Alvarez T D, St. George M, Appelbaum L G, Bellugi U, Neville H 2000 Electrophysiological studies of face processing in Williams syndrome. *Journal of Cognitive Neuroscience* **12**(11): 47–64
- Pani J R, Mervis C B, Robinson B F 1999 Global spatial organization by individuals with Williams syndrome. *Psychological Science* **10**(5): 453–8
- Paterson S J, Brown J H, Gsodl M K, Johnson M H, Karmiloff-Smith A 1999 Cognitive modularity and genetic disorders. *Science* **286**(5448): 2355–8
- Pinker S 1994 *The Language Instinct*. Penguin, Harmondsworth, UK
- Pinker S 1999 *Words and Rules*. Weidenfeld & Nicolson, London
- Pober B R, Dykens E M 1996 Williams syndrome: An overview of medical cognitive and behavioural features. *Mental Retardation* **5**: 929–43
- Rae C, Karmiloff-Smith A, Lee M A, Dixon R M, Grant J, Blamire A M, Thompson C H, Styles P, Radda G K 1998 Brain biochemistry in Williams syndrome: Evidence for a role of the cerebellum in cognition? *Neurology* **51**: 33–40
- Rossen M, Klima E S, Bellugi U, Bihrlé A, Jones W 1996 Interaction between language and cognition: Evidence from Williams syndrome. In: Beitchman J H, Cohen J H,

- Konstantareas M M, Tannock R (eds.) *Language Learning and Behavior Disorders: Developmental Biological and Clinical Perspectives*. Cambridge University Press, New York, pp. 367–92
- Tager-Flusberg H, Boshart J, Baron-Cohen S 1998 Reading the windows to the soul: Evidence of domain-specific sparing in Williams syndrome. *Journal of Cognitive Neuroscience* **10**: 631–9
- Tassabehji M, Metcalfe K, Fergusson W D, Carette M J A, Dore J K, Donnai D, Read A P, Proschel C, Gutowski N J, Mao X, Sheer D 1996 LIM-kinase deleted in Williams syndrome. *Nature Genetics* **13**: 272–3
- Tassabehji M, Metcalfe K, Karmiloff-Smith A, Carette M J, Grant J, Dennis N, Reardon W, Splitt M, Read A P, Donnai D 1999 Williams syndrome: Use of chromosomal microdeletions as a tool to dissect cognitive and physical phenotypes. *American Journal of Human Genetics* **64**: 118–25
- Temple C M 1997 Cognitive neuropsychology and its applications to children. *Journal of Child Psychology and Psychiatry* **38**: 27–52
- Thomas M S C, Grant J, Gsödl M, Laing E, Barham Z, Lakusta L, Tyler L K, Grice S, Paterson S, Karmiloff-Smith A 2001 Can atypical phenotypes be used to fractionate the language system? The case of Williams syndrome. *Language and Cognitive Processes* **16**(2/3): 143–76
- Udwin O, Yule W 1991 A cognitive and behavioural phenotype in Williams syndrome. *Journal of Clinical and Experimental Neuropsychology* **13**: 232–44
- Vicari S, Carlesimo G, Brizzolara D, Pezzini G 1996 Short-term memory in children with Williams syndrome: A reduced contribution of lexical-semantic knowledge to word span. *Neuropsychologia* **34**: 919–25
- Vicari S, Bellucci S, Carlesimo G A 2001 Procedural learning deficit in children with Williams syndrome. *Neuropsychologia* **39**(7): 665–77
- Volterra V, Capirci O, Pezzini G, Sabbadini L, Vicari S 1996 Linguistic abilities in Italian children with Williams syndrome. *Cortex* **32**: 663–77
- Wilson B, Cockburn J, Baddeley A 1985 *Rivermead Behavioural Memory Test*. Thames Valley Test Co., Reading, UK

A. Karmiloff-Smith

Wisdom: Philosophical Aspects

Wisdom is not a central topic of contemporary philosophy. Today's philosophers seem to confirm Nietzsche's observation: 'When philosophers meet among themselves they start casting off all sorts of wonderful rubbish; above all ... they hang up "the love of wisdom" like stuffy robes of office' (Nietzsche 1980a, p. 511). Common sense, on the contrary, still expects wisdom to be provided by philosophy, and the discipline's social reputation is partly based on this expectation. But as contemporary philosophy seems scarcely inclined or able to fulfill this demand, common sense draws on forms of wisdom from non-occidental cultures or from esoterics. While 'wisdom' is a term rarely used in contemporary philosophy, the

literature about wisdom from the esoteric and the anthropological-cultural side is flourishing.

There is nothing wrong with sensing deficiencies in the academic field and turning to other sources instead. Yet in the second part of this article I will demonstrate that contemporary philosophy can in fact provide a great many elements of wisdom appropriate for our times. The first part is devoted to an accurate reconstruction of the main historical turns in the philosophical understanding of wisdom.

1. Philosophy—A Concept Introduced by Plato

As is commonly known, philosophy is related through its name to wisdom: 'philo-sophy' (in Greek: *philo-sophia*) means 'love of wisdom' or 'quest for wisdom' (in Greek: *sophia*). But this concept of *philosophia* corresponds neither to the original understanding of philosophical thinking nor to that of wisdom. The concept of *philosophia* was first created by Plato in the first half of the fourth century BC.

1.1 The Pre-Platonic Understanding of Philosophy and Wisdom

Philosophers before Plato neither deployed the term 'philosophy' nor described their enterprise with respect to wisdom (*sophia*). Rather, they characterized their doctrines as 'histories'. Moreover, the Greek understanding of wisdom—as paradigmatically expressed in the topical talk of the 'Seven Sages of Greece'—focused on practical, not theoretical knowledge. In everyday language, a wise person was just an expert of some kind or other (a craftsman, artist, doctor, military leader etc.); in order to be included in the catalogue of the Seven Sages his work had, in addition, to be of extraordinary political relevance. So Thales, the only ancient philosopher ever to figure among the Seven Sages, was included in this group not as a philosopher, but as an astronomer and engineer who brought military success to his community by predicting an eclipse and redirecting a river.

1.2 Plato's Turn

What, then, is the specific sense of Plato's introduction of the concept of *philo-sophia*—an innovation so successful that his concept is commonly taken to be the concept of philosophy altogether?

Three shifts are characteristic. First, the emphasis in the understanding of *sophia* as well as *philosophia* is put on theory instead of praxis. Philosophers observe the nature of things not for the sake of practical usage but for that of pure cognition. The new concept of philosophy is strictly opposed to the old practical aims of *sophia*. Second, a border line is drawn between

16504