Towards understanding atypical social affiliation in psychopathy

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Summary

One hallmark of individuals with psychopathy is their reduced motivation and capacity to develop authentic social relationships, founded on an enjoyment of prosocial interactions or concern for others. Surprisingly, potential neurocognitive vulnerabilities contributing to atypical social affiliation and lack of prosocial behaviours in psychopathy have yet to be systematically investigated. To date research efforts have largely focused on how individuals with psychopathy process negative emotions and how this may impact their capacity to empathise with others' distress or feel guilt. Here we propose a framework for understanding the development of atypical social affiliation and attachment in psychopathy, and outline several key processes and neural systems understood to underpin them. We then describe current neurocognitive findings that suggest that these are compromised in individuals with or at risk of developing psychopathy. Finally, we consider a number of research directions that would help shed light on the aetiology and development of social affiliation in psychopathy and argue that this line of work has the potential to inform and enhance prevention and treatment strategies.

Introduction

Individuals with psychopathy are characterized by lack of empathy and remorse, manipulation of other people, the ability to engage in premeditated, cold and calculated aggression to achieve their goals and impoverished capacity to take care of their responsibilities and make good decisions.[1,2] The extant evidence base suggests that heritable individual differences account for a substantial proportion of variation associated with risk of developing psychopathy.[3] Decades of experimental and neurocognitive research have focused on understanding why individuals with psychopathy (or those at risk of developing the disorder) do not readily empathise with other people's distress or why they make poor decisions. These studies have demonstrated atypical structure and function in a network of emotion and reward processing areas that are thought to facilitate emotional resonance, empathy, and decision making guided by reinforcement information.[4–6] Prior studies have also demonstrated intact ability to take the perspective of other people in individuals with high levels of psychopathic features,[8,9] although new work suggests that they may be less likely to do so spontaneously.[10] Collectively this work has considerably advanced our understanding of the neurocognitive presentation that accompanies psychopathic features and helps explain why indivdiuals with these features appear relatively unaffected by other people's distress, are able to manipulate and deceive others when they are motivated to do so, can commit calculated acts of aggression against other people, and often make repeated disadvantageous decisions.

By contrast what leads to atypical social affiliation and lack of prosocial behaviours in psychopathy has received less attention. A particular hallmark of individuals with psychopathy is their reduced motivation and capacity to develop social relationships founded on an enjoyment of prosocial interaction or genuine love and concern for others' well-being.[1] They are also characterised by 'looking after number one', not fulfilling their responsibilities and behaving in ways that violate the rights of other people – behaviours that are antithetic to the prosocial and caring behaviours that most of us regularly engage in.[1] Work by Lynam and colleagues has demonstrated that low communion/antagonism are core features of

psychopathy; moreover, individuals with such features show attenuated prosocial responses even to those with whom they have close relationships.[11,12,13] Yet, remarkably, there has been very little work to date investigating potential underlying neurocognitive mechanisms of impaired affiliation and social connectedness in individuals with high levels of psychopathic traits. Many of the neurocognitive mechanisms that subserve empathy for distress, may also be involved in processing positive affective/affiliative cues that promote social cohesion and bonding. What is outstanding is a systematic investigation of how these neurocognitive mechanisms (and others) are implicated in development of psychopathy in the context of stimuli that promotes affiliation and prosocial engagement. Advancing this line of research will be of theoretical interest, as it has the potential to provide a more complete characterisation of psychopathic presentation. Work in this area also has the potential to be of significant clinical relevance as many clinical programmes aimed at reducing antisocial behavior and promoting prosocial functioning rely on building relationships.[14]

In this paper we will propose a framework for understanding the development of atypical affiliation and attachment in individuals with psychopathy. We will outline some key processes and neural systems understood to support attachment and affiliation, and describe current neurocognitive findings that suggest that these are compromised in individuals with or at risk of developing the condition. We will then outline how systematic research into the development of affiliation/attachment patterns that characterise psychopathy, as well as their aetiological origins, has the potential to inform and enhance prevention and treatment strategies.

Affiliation and attachment in psychopathy

All group-living animals have a basic drive to affiliate with conspecifics.[12] Humans are not an exception. We are intrinsically social animals, typically forming enduring affiliative/attachment bonds with others.[15,16] We regularly meet the needs of others (and have our needs met), particularly needs of those who are considered part of our 'in-group'.

Depue and Morrone-Strupinsky have proposed that there are substantial individual differences in human trait affiliation, underpinned by differences in neurobiology.[17] One of the striking characteristics of individuals with psychopathy (or those at risk of developing the condition) is that their relationships seem shallow, transient and transactional, with several studies indicating that they show reduced quality of attachment, peer, romantic and work relationships. For example, studies have reported an association between psychopathic features and disorganized attachment, [18,19] peer relationships characterized by less stability and greater conflict in children,[20] and poor quality of marital relationships in adults.[21] Individuals with high levels of psychopathic features are also rated as being less reliable team players in the work place.[22] They appear less inclined to ingratiate themselves with other people, unless there is clear self-interest to do so.[23]

Cognitive processes in affiliation and attachment

Humans' first attachment/affiliative bonds are constructed in an interactive fashion with their caregivers, with both the caregiver and the child contributing to the quality and the nature of the relationship that emerges between them.[15,24,25] From infancy onwards, children learn about their environment via pairing of their bodily sensations with visual, auditory and tactile cues that the caregiver mirrors back to them.[15,24] Different caregivers may be more or less reliable in helping the child form accurate contingencies; equally there are individual differences in the propensity of children to detect, elicit and process cues from the environment, for example individual differences in following the gaze of their caregiver. Such reciprocal interactions when contingently aligned create what is known as biobehavioural synchrony, a critical process in consolidating attachment/affiliative bonds, promoting survival and related collaborative goals.[15,26] Biobehavioural synchrony manifests itself in mirrored behaviours, synchronized autonomic responses, coordinated hormonal release and coupling of brain responses in the key

nodes of the social brain network between humans who interact together. In the early years the relationship with the caregiver is the primary sphere where the patterns of affiliative behaviour are established.

The human tendency to affiliate later widens to friends, romantic (and even academic) partners and the wider community and is in part initiated and maintained by positive affective signals and affective resonance with other people. For example, genuine laughter is a universal expression of positive affect used to maintain social bonds in humans and in animals.[27–30] It is a highly contagious behaviour: it can be primed simply by listening to others' laughter.[31] Such emotional contagion has been posited as a mechanism for facilitating the coupling of emotions and behaviour within groups, increasing cooperation, cohesiveness, and social connectedness.[27,28,32] Genuine laughter also plays a role in the vicarious experience of positive emotions, and it triggers the endogenous opioid system, argued to be key for prosocial communication and social bonding in primates and other mammals.[28,33,34] Attending to and understanding other minds and deciphering people's affective states and motivations, mentalising, is thought to be a further important aspect of affiliative /attachment relationships.[15,26,35,36] In order to integrate socially, humans need to be able to appreciate that other people's perspectives and goals may differ from their own and must process and keep in mind the preferences and concerns of those that they interact with.

Neural systems in affiliation and attachment

The neurobiology of human attachments relies on a network of brain regions processing reward, motivation, salience, learning, memory, interoception and mentalising (see [15] and [17]for excellent reviews). Attachment-related motivational behaviours such as social orienting and social seeking (e.g. following eye-gaze), as well as maintaining contact across extended periods are proposed to depend on the brain's 'reward-motivation' system. This system includes the striatum, amygdala, ventral tegmental area, orbitofrontal cortex, ventromedial prefrontal cortex

and anterior cingulate. The ability to join in and resonate with other people's emotions (such as genuine laughter) is also critical for maintaining affiliative relationships and is thought to depend on the 'empathy network' (the same network that is also implicated in empathic response to others' distress), including the insula, anterior cingulate cortex, inferior frontal gyrus and supplementary motor area. Finally, the so called 'mentalising' network is proposed to be critical for integrating resonance with other people with understanding of their motives and goals, thus supporting attachment formation. This network includes superior temporal sulcus, posterior cingulate cortex, temporo-parietal junction and medial prefrontal cortex.

Psychopathy: Cognitive and neural function in domains important for affiliation and attachment

Experimental studies of adult individuals with psychopathy and children at risk of developing the condition have reported atypical functioning in a range of processes and associated neural regions implicated in affiliation and attachment (note these processes are also involved in supporting other behaviours). Here we examine three areas of social processing, including eye-gaze/orienting to others, laughter, and mentalising. Research in each of these areas has begun to shed light on disrupted affiliative functioning in individuals with or at risk of developing psychopathy.

Eye gaze: Studies by Dadds and colleagues were the first to demonstrate that children and adolescents with high levels of psychopathic features (specifically callous-unemotional aspects of psychopathic personality) make less eye contact with their mothers than their peers, in both free play and directed situations.[37–39] Their findings indicated that the reduced eye-gaze was driven by the child - the mothers of children with high levels of psychopathic features did not differ from other mothers in the amount of eye contact they attempted to make with their children. More recently, Bedford and colleagues have conducted prospective longitudinal analyses focusing on 5-week-old infants' preferential orientation to the human face (vs. an inanimate stimulus) and subsequent development of psychopathic (callous-unemotional)

features in early childhood.[40] Reduced tracking of the mother's face by an infant was associated with development of subsequent callous-unemotional features at the age of 2.5 years. Another study by the same lead author (focusing on a different sample of older babies (6 months) and utilizing another face orienting measure) showed an interaction between face orienting and maternal sensitivity, such that those babies with lowest orientation preferences and lowest maternal sensitivity were the most likely to develop subsequent psychopathic features.[41] No brain imaging study to date has specifically focused on face orienting in individuals with or at risk of developing psychopathy. However, many structural and functional imaging studies implicate the 'reward-motivation' networks, thought to be critical for biobehavioural synchrony, in the pathophysiology of psychopathy.[5,42] For example, adults with psychopathy and children at risk of developing psychopathy show reduced amygdala activity to salient social stimuli, including other people's distress emotions. A single study of children at risk of developing psychopathy further indicated that deliberate orienting of attention to critical feature of distressed faces (eyes), does not boost amygdala functioning to this salient social stimuli in children at risk of developing psychopathy.[43]

Laughter: A recent study by our research group is the only investigation to date to focus on how children at risk of developing psychopathy process genuine laughter.[44] We asked the children to listen to clips of genuine laughter in the scanner. After the scanning session they listened to the laugher stimuli again and reported their desire to join in with the laughter. Neuroimaging studies of typical individuals demonstrate that listening to laughter automatically recruits motor and premotor regions involved in the production of emotional expressions and empathy[26] including the precentral gyrus, supplementary motor area, inferior frontal gyrus, and anterior insula.[32,45-47] The preparatory motor response associated with laughter production is thought to facilitate joining in with others' positive vocalizations during social behaviour, representing a neural mechanism for experiencing these emotions vicariously and promoting social connectedness.[28,32] Compared with typically developing boys, those at risk of developing

psychopathy displayed reduced neural response to genuine laughter in the supplementary motor area and anterior insula. This finding indicates basic difference in how children at risk of developing psychopathy respond to genuine laughter. They also reported reduced desire to join in with others' genuine laughter, compared with their typically developing peers matched on ability and socioeconomic status. The reduced anterior insula response in part accounted for the reduced desire to join in with others' laughter in the group at risk of developing psychopathy. These findings open a new and interesting avenue of research to explore the role of positive affective signals that facilitate social affiliation, and promote and maintain social bonds, in individuals with or at risk of developing psychopathy. In light of the fact that individuals with psychopathy are high on antagonism,[11,12,13] one possibility is that they less readily experience positive affect that promotes resonating with other people's laughter and joining in. This line of enquiry will add to the more established evidence base regarding the atypical development of empathy for distress in this population.

Mentalising: One of the defining features of individuals with psychopathy or those at risk of developing the disorder is their ability to successfully manipulate their victims. Consistent with this observation, several studies that measure mentalising (alternatively, cognitive perspective taking or theory of mind) - without a requirement to process affective information - report no impairments in adults and children with psychopathic features on this domain.[8,48–53] Research from our group has also shown that when children at risk of developing psychopathy engage in mentalising computations that do not have affective content, they do not differ from typically developing individuals in how they recruit critical nodes of the mentalising network in the brain, such as the medial prefrontal cortex and temporoparietal junction.[9] Collectively, these findings contrast with those reported for individuals on the autism spectrum, for whom difficulties in mentalising represent a core feature of the condition. A recent study that focused on the *propensity*, rather than ability, to take other people's perspective, found an impairment in adults with psychopathy.[10] In other words, the picture that is emerging from the experimental

evidence is that individuals with or at risk of developing psychopathy have the capacity to mentalise, but may not share the propensity to do so as readily as other people. This area warrants further investigation and it would be critical to elucidate why individuals with psychopathy think about other people's minds only when explicitly required to do so or when it directly benefits them.

The cognitive-affective functions reviewed above, and the neural networks that support them, are thought to be important for attachment formation and for sustaining affiliative relationships. The extant evidence indicates that individuals with or at risk of developing psychopathy present with altered functioning in each of these domains in ways that may compromise their capacity and propensity for affiliation and prosocial behaviour. While these preliminary findings are of interest they also require replication. Moreover, a wider set of experimental tasks is needed in order to investigate these functions, as well as other potential neurocognitive mechanisms that may underlie atypical social affiliation in psychopathy. Most critically we need longitudinal data capable of shedding light on issues of causality and how affiliative relationships are formed during development. For example, we do not currently have data on how disruptions in tracking a caregiver's face might shape development of emotion recognition in children at risk of developing psychopathy. We also do not know whether the attenuated behavioural and neural response to laughter is a robust indicator of dispositional risk for psychopathy and to what extent it represents a cause versus a consequence of atypical social development. Finally, there currently are no data to elucidate why individuals with psychopathy have an intact ability to mentalise (which they can effectively deploy to manipulate other people), yet show a diminished propensity to consider and be interested in other minds. Longitudinal studies designed to elucidate both the impact of the social environment on an individual, as well as the impact of an individual on shaping their social environment, will be critical for understanding formation of psychopathic risk across development. Such work needs to be undertaken within a multi-level framework that considers brain maturation in the context

of biological and environmental vulnerabilities, and focus on the transactional nature of social development. We will discuss next future research needs in more detail and also outline the potential translational implications of research into social affiliation and attachment in psychopathy.

Future Research Needs

In order to advance our understanding of the developmental risk for psychopathy, we suggest two broad lines of enquiry.

First, what are the aetiological origins of disrupted social affiliation in psychopathy? Psychopathic features in children are moderately to strongly heritable.[42] There is also tentative evidence that antisocial behaviour in the presence of psychopathic features may be more strongly heritable than antisocial behaviour in the absence of psychopathic features.[54,55] However, it is critical to emphasise that even a high heritability estimate does not denote genetic destiny. Any heritability estimate reflects the impact of genetic vs. environmental influences on individual differences or group differences. There is always a degree of error in the estimates (they are not precise), the relative proportion of genetic vs. environmental influences may differ between populations, and any estimates do not tell us anything about the origins of psychopathic features for a specific, single individual. Furthermore, there are no genes for psychopathy. This may sound a bizarre claim as we have just said that psychopathic features are heritable, but the way that risk genes for psychopathy operate is probabilistic, rather than deterministic: genes do not code for psychopathy. Genes code for proteins that influence characteristics such as neurocognitive vulnerabilities that may in turn increase risk for developing psychopathy, particularly under certain environmental conditions.

In order to address the question of the aetiological origins of disrupted social affiliation in psychopathy, we need to conduct new genetically informative studies. Heritability estimates

indicate the sum total of genetic influences on risk of developing psychopathy, but do not identify the genes involved and have not specifically focused on atypical social affiliation in psychopathy. We know from animal work that dopamine orients individuals to primary rewards like food and sex; whilst orienting to social reward (e.g. conspecifics, including the primary caregiver) depends on co-activation of dopamine and oxytocin systems.[15] Oxytocin calibrates arousal to and orienting to social stimuli and phasic co-activation with dopamine imbues the central social interactions with a robust reward value. This in turn helps maintain orientation to socially relevant stimuli and promotes biobehavioural synchrony over development. Development of brain networks that support affiliative and attachment behaviours happens in response to dopamine and oxytocin inputs over time.[15] Work with humans, e.g. as specified by the Social Salience Hypothesis of Oxytocin, is also indicating an important role for oxytocin in affiliating with in-group members and promoting prosocial behaviours.[56] There is tentative evidence that developmental risk for psychopathy may be associated with genetic variants that predispose to attenuated functioning of the oxytocin system, although this work requires replication and extension to large study populations.[42,57-59] If, in the future, we were able to obtain polygenic risk scores that relate to functioning of systems important for supporting development of social affiliation (e.g. oxytocin release and regulation), these could be used in longitudinal samples followed up from infancy. This would enable scientists to test, for example, whether infants with high polygenic risk scores have significant disruptions to the early development of biobehavioural synchrony - as indexed by standardised experimental measures shortly after birth. It would also enable scientists to ask targeted questions about gene-environment interplay and how it unfolds. Do, for example, infants at genetic risk (as measured by a polygenic risk score) shape parental responses and impact the formation of biobehavioural synchrony in a different way than their peers with low genetic risk? In other words, does evocative gene-environment correlation impact the trajectory of biobehavioural synchrony development? Researchers could also investigate whether particular parenting behaviours or other environmental factors exacerbate elevated genetic risk

by impacting the trajectory of biobehavioural synchrony development? This could be most conclusively addressed in the context of infants who have been adopted away at birth, as in this case the researchers could be confident that they are measuring gene-environment interaction, rather than gene-environment correlation.

Finally, it is important to note that a number of recent studies suggest that there are individuals who present with callous-unemotional psychopathic features following extreme childhood adversity (See [60] for a summary). We can think of them as 'behavioural phenocopy' of primary psychopathy, but with a distinct developmental route to a psychopathic behavioural profile. These individuals appear callous and uncaring and display high levels of antisocial behaviour, but in contrast to primary psychopaths display internalizing problems and a distinct neurocognitive profile that is in line with extreme threat reactivity.[60-62] We need to investigate neurocognitive processes related to social affiliation in this group and how their social affiliative behaviours develop. In other words, we must be open to systematically investigating different developmental pathways into psychopathic presentation.

Second, to what degree does disrupted biobehavioural synchrony play a causal role in the emergence of atypical social affiliative behaviour and what are the underlying neurocognitive processes that mediate this relationship across development?

We know from how individuals with or at risk of developing psychopathy behave, including how they behave in some experimentally controlled tasks. We know that positive, affiliative emotions do not seem to have the same motivational value for them and that other people's emotions, thoughts and needs are not automatically attended to or prioritised. We can speculate that atypical biobehavioural synchrony may have a knock-on effect over development, where brain circuits responsible for initiating and maintaining affiliation / attachment are not calibrated in a normative way in individuals who go on to develop psychopathy. There is

tentative evidence that there may be disruption of biobehavioural synchrony, driven by the child, or both the child and the parent (a 'double hazard'), in infants at risk of developing psychopathic features.[40,41] Atypical biobehavioural synchrony could thus index the degree to which positive affiliative signals are inherently rewarding or have potential to become socially rewarding over development, or whether a child has the propensity to processes other minds later in life.

We propose, based on developmental literature, that neural systems supporting mentalising computations come on-line at around age of four and do so in a normal fashion for individuals at risk for developing psychopathy. However, social motivational value of conspecifics, as calibrated by development of biobehavioural synchrony, may influence how readily someone engages in spontaneously mentalising about others. If others are not inherently rewarding, but only hold reward value insomuch as they can represent an instrumental gain, then a person might only engage in mentalising about others when they need something from them – which is what we see in the case of individuals with or at risk of developing psychopathy. In other words, the value of other people's needs to you would determine how readily you mentalise with them. To test this proposal, we need longitudinal studies that include measures of biobehavioural synchrony in infancy and subsequent measures relating to social affiliation / prosocial behaviour. These could include: a) standardised rating scale, interview or observational measures indexing development of social affiliation and prosocial behaviour; and b) experimental measures charting neurocognitive processes thought to be critical for social affiliation – including, for example, processing of laughter and propensity to mentalise.

Translational implications

Psychopathy incurs significant financial and human costs for society. Current treatment approaches for adults with psychopathy have modest effectiveness.[63] The evidence for efficacy of treatment for children and young people at risk of developing psychopathy is mixed, but

more hopeful.[64-66] We want to raise two important considerations in relation to prevention and treatment in the context of children at risk of developing psychopathy. First, we propose that it is not sufficient to focus on behaviour modification and teaching caregivers and teachers behaviour management techniques. It is also important to understand why it can be more challenging to deliver traditional systemic approaches with this population. A conceptual framework is needed that that can help to inform alternative interpretations of behavioural difficulty and motivate the rationale for effective support for those caring for these children. Second, we outline a number of reasons why it may be particularly fruitful to develop techniques for motivating affiliative, prosocial behaviour in children at risk.

Interventions for children with conduct problems (including those at risk of developing psychopathy) predominantly draw on systemic principles, focusing on the relationship between the child, their peers and the adults around them (e.g. parents, carers, teachers, social workers). Yet, many aspects of establishing a mutual and balanced reciprocal relationship are contingent on prosocial and affiliative processes that function quite differently in children at risk for psychopathy. Atypical affect processing and a reduced drive to affiliate with others is likely to contribute to a distinct pattern of socialisation difficulties. Currently we have a poor understanding of precisely how such atypical affiliative processing and behaviour could inform the formulation of a child's presenting problems and guide approaches to change. The substantial variability in how children with conduct problems respond to interventions may in part derive from the impact of these specific information processing biases in how they process social/affiliative stimuli.

Social learning principles used in therapeutic programmes emphasise the ways in which adult behaviour can impact on the child outcome. However, children also play a key role in shaping the responses of adults around them, and in this case often evoking particularly negative reactions. Furthermore, parents of these children may share some of the vulnerabilities of their

child, augmenting the challenge of delivering a systemic intervention. Helping parents, carers and teachers reframe a child's behaviour (including in relation to affiliative behaviour) in the context of a profile of dispositional strengths and weaknesses that the child presents with may change how the adults around them experience and respond. Moreover, having systems in place to ensure that adults caring for the child themselves receive support and a space to process their relationship with the child is a prerequisite for providing sustained, predictable and support.

A more precise understanding the neurocognitive processes that contribute to the atypical affiliation could help sharpen the clinical formulation. Some previous work with adults with and children at risk of developing psychopathy has focused on lack of empathy (as opposed to affiliation) and trialed effortful strategies to modify how negative/fearful stimuli are processed. For example, participants have been asked to upregulate their emotional response or direct attention to the relevant features of the face. [67,68] While neurocognitive changes towards more typical presentation have been observed in these laboratory studies, there is no empirical or clinical evidence that individuals with psychopathic features are then motivated to apply such strategies in everyday life. This is consistent with the long recognized persistent deficits in victim empathy in this population.[69] Consequently, exploiting low-level automatic processes, such as conditioning and attentional bias modification, may represent more effective ways to modify behaviour. However, modification of how negative stimuli (such as another's pain or distress) are processed using implicit strategies is inherently ethically problematic. It is not at all clear that aversive conditioning or attentional cuing to such stimuli would engender victim empathy and elicit desired behavioural outcomes; indeed, utilising such approaches may simply produce heightened arousal and behavioural unpredictability. By contrast, promoting responses to positive affect by modifying automatic/implicit processing – such as by pairing social affiliative stimuli with stimuli that the child finds rewarding - has the potential to make a child

more receptive to adult affect/feedback/behaviour modification, thus offering a potential to scaffold existing intervention approaches.

Conclusion

One way to understand the atypical pattern of social behaviour we see in individuals with psychopathy is to imagine that they are essentially looking out for an extremely restricted 'in-group': themselves. Various affiliation and attachment related cognitions no doubt evolved to promote mutualistic social investment and collaboration within groups.[70] Atypical functioning of the neurocognitive systems that give rise to these cognitions could lead to an alternative adaptive strategy that involves promotion of oneself at others' expense, particularly if the other person's distress is also not experienced as salient or aversive.[5,71-73] We know that individuals with psychopathy lack typical affiliative relationships and have little altruistic concern for others. We need to better understand why and how affiliation and attachment 'derails' in these individuals over development and how it relates to the development of particular aspects of psychopathic personality (e.g. callous-unemotional traits). Work in this area has the potential to yield crucial information that can be used to promote prosocial functioning in individuals who are not able to arrive at that outcome via ordinary means, with serious consequences for themselves and society.

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Search Strategy and Selection Criteria

References for this review were identified through searches of PubMed by the use of terms “psychopathy” and “callous-unemotional” with one of the following: “affiliation”, “attachment”, “orienting’, “laughter”, “mentalising”, “theory of mind”, “positive affect”, “emotion”.

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