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IS THE MORNING-AFTER PILL LEVONORGESTREL A CONTRACEPTIVE OR AN ABORTIFACIENT? OR BOTH?

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SUMMARY POINTS

- The US Food and Drug Administration states explicitly that levonorgestrel (LNG) does not cause an abortion.
- Patient information for the two brands of emergency contraception available in the UK do not indicate that an abortion can take place.
- There are studies which conclude that LNG causes embryos to die in a significant minority or even a majority of cases.
- The precise way in which LNG works is still not certain.
- It can act as both a contraception or an abortifacient as it has different effects when taken before or after ovulation.
- Different ways LNG can act:
    - LNG does not seem to affect sperm or the process of fertilisation.
    - The majority of studies show that ovulation is delayed or suppressed if LNG is taken four days before ovulation or earlier, suggesting a contraceptive action has taken place.
    - Taken two or three days before ovulation, LNG is largely incapable of preventing ovulation. So, if fertilisation has occurred LNG could have an abortifacient action.
    - There is evidence that LNG may reduce the movement of the embryo in the fallopian tube. Although not proven, this suggests that embryos conceived after LNG could be prevented from implantation due to slow movement through the fallopian tube.
    - LNG does not appear to prevent embryo implantation and development, when taken on the day of or after ovulation.
    - LNG taken before ovulation can interfere with the processes which prepare the lining of the womb. The embryo might arrive and be unable to attach to the lining of the womb and will, therefore, die. LNG, taken at this stage, may also cause premature shedding of the endometrium, causing the embryo to die when it reaches the womb.

INTRODUCTION

Levonorgestrel (LNG) is one of several agents that are marketed as emergency contraception (EC) for use up to 3 days after unprotected intercourse or contraceptive failure. LNG is a progestin, also referred to as a progestogen, and it primarily activates progesterone receptors. That is, it is effectively an artificial progesterone, but with some other actions as well.

Levonelle One Step, which contains LNG, and ellaOne, in which the active ingredient is ulipristal, are the two main emergency contraception brands available in the UK, and the IUD to a lesser extent. The patient information for LNF and Ulipristal describes them as solely contraceptive and not abortifacient.[1,2]

While it is generally accepted that the IUD can cause the demise of an early embryo, and the evidence for ulipristal acting similarly is strong,[3] there has been much controversy and uncertainty about the mode of action of LNG. Does it act solely by stopping ovulation, or at least delaying it sufficiently to make fertilisation impossible, or can it abort the newly conceived embryo that would otherwise implant in the uterine lining and continue developing?

This answer is obviously important information for women. It ensures informed consent, and some women will refuse to use LNG if they know it could be abortifacient. This is the concern expressed in a 2022 widely cited review of the mode of action of LNG. Endler *et al.* note, "Misconceptions regarding its mechanism of action contribute to low use in some settings."[4] In other words, some women are not using it because they are worried they might be aborting their embryo. The review starts with the definitive statement that LNG " … inhibits ovulation to prevent fertilisation",[5] before going on to look at the research, which the authors interpret as confirming their view. But beyond the need for scientific accuracy to enable informed consent, in the US post-*Dobbs* environment, some see another problem – states that have restricted abortions might also regulate LNG more tightly if it was known to have an abortifacient effect. As Adashi *et al.* note,

> The mechanism of action of levonorgestrel has long been a matter of concern, both politically and scientifically. Does levonorgestrel prevent fertilization, or does it interrupt development after fertilization? The political implications of this question have been sharpened by the US Supreme Court decision in *Dobbs v Jackson Women's Health Organization*. With the removal of legal protections for access to abortion, drugs that act as abortifacients are themselves under threat. The question of whether levonorgestrel might act as an abortifacient thus becomes critical.[6]

Following publication of the Endler *et al.* review in May 2022, the *US Food and Drug Administration* (FDA) agreed in late December 2022 to alter the product label and consumer information regarding LNG to explicitly state that it "does not terminate pregnancy".[7,8] The FDA made this decision in response to an application by the maker of LNG, *Foundation Consumer Healthcare* (FCH). In describing the change and their role in achieving it, FCH refer to the Endler *et al.* review, using it to claim that the "Scientific data overwhelmingly shows that Plan B [LNG] works by delaying or stopping the release of an egg from the ovary and will not prevent or interfere with the implantation of a fertilized egg."[9] It is not surprising that FCH refer to this review and no other – they provided funding for it, and it provided the answer they wanted. The review's authors have also written extensively in support of abortion and the means to achieve it, and state in their

declaration of competing interests that they are invited speakers or advisors for many drug companies that manufacture or market abortion drugs or abortifacient ones.[10] The declaration does not detail any remuneration involved, but the conflicts of interest are clear.

Is the review and the FDA declaration the final word on the mode of action of LNG? Given the complexity of the processes involved and the research limitations, how can FCH, Endler *et al.*, and the FDA give such a definitive answer? At this point in time, critical pieces of information like the time of ovulation or whether conception has or has not occurred cannot be known with precision. Uncertainty makes decision-making difficult, but if there really is uncertainty, then it is the task of science, its interpreters, and the medical authorities to be transparent about that.

Contrary to the FDA and the conclusion of other medical authorities,[11] and the review by Endler *et al.* (that LNG works *only* by inhibiting or delaying ovulation), several other reviews instead conclude that LNG causes embryo demise in a significant minority or possibly even majority of cases.[12,13,14,15,16,17] The FDA and others clearly do not agree with these authors' conclusions.

This paper will look at the evidence in some detail. But first it will provide a very basic description of the physiological processes involved to assist a better understanding of the stages and sites at which LNG might conceivably act.

REPRODUCTIVE PHYSIOLOGY

The female cycle is controlled by hormones from the pituitary gland; *Follicle Stimulating Hormone* (FSH) and *Luteinizing Hormone* (LH), and by hormones from the ovaries, *Estrogen and Progesterone*. FSH stimulates the development of the ovarian follicles, each of which contains an egg (ovum). As the follicles develop, they release estrogen, and as its level increases it feeds back to the pituitary to decrease FSH until only one dominant follicle is supported in its development. Increasing estrogen also causes the endometrium to build up in preparation for embryo implantation and continuing pregnancy and causes changes to the cervical mucus that assist sperm passage.

The increase in estrogen is followed by its decline, which appears to have a role in triggering the release of LH from the pituitary, leading to what is commonly called the LH surge, which leads to maturation of the dominant follicle and then ovulation. The onset of the LH surge occurs some 36 hours before ovulation, and peaks about 12-24 hours before ovulation. If fertilisation occurs, it does so in the fallopian tube, after which the embryo travels toward the uterus, implanting in the endometrial lining about one week after fertilisation.

The remnant of the ovarian follicle, now termed the corpus luteum, releases progesterone that, along with estrogen, encourages further preparation of the endometrium to support implantation and ongoing embryonic development.

At around the time of implantation (approximately 1 week after conception), cells from the embryo release *Human Chorionic Gonadotropin* (HCG), which further stimulates the corpus luteum to produce progesterone and estrogen. HCG measurement is used in pregnancy tests to confirm what is often called a clinical pregnancy. However, it is important to be clear that pregnancy starts with fertilisation, but there is no test yet available to determine that moment.

The phase of the cycle prior to ovulation is termed the pre-ovulatory, follicular, or proliferative phase (referring to endometrial development); and after ovulation, as the post-ovulatory, luteal or secretory phase (referring to endometrial secretions).

There are also characteristics of sperm that are related to fertility, as well as variations in the microenvironment around sperm that affect their ability to reach and fertilise the egg. Once fertilisation has occurred, there is 'cross-talk' between the embryo and the environment of the fallopian tube and uterus. Little is known about these interactions, but studies have identified some of the agents involved.[18]

Other characteristics of this process that are important for understanding what LNG might do include the fact that sperm may survive for about 5 days in the reproductive tract and the egg can survive for about 24 hours after ovulation. Hence, there is a 'fertile window' during which fertilisation/conception is possible. In broad terms, this window is typically accepted as the five days before ovulation and the day of ovulation itself. This yields 6 fertile days denoted -5 through to 0, the day of ovulation.

As noted, there is currently no reliable measure of the moment of ovulation or the moment of fertilisation. The best that can be done about ovulation is to measure LH levels as well as other hormones to gain an approximation within about 24-48 hours. Alternatively, transvaginal ultrasound (TVUS) can measure follicular diameter to obtain an assessment of the likelihood of follicular rupture and egg release. TVUS can also indicate that ovulation has occurred by observing the presence of the corpus luteum.

This brief description does not do justice to the complexity involved in enabling conception and the ongoing development of the human embryo. There are other biochemical agents, feedback loops, gene regulators, and endometrial receptivity markers that control a variety of steps in what is a critically timed sequence of events, and research is ongoing. Importantly, disruption of any particular stage or process at a given point in time may not only affect that process at that time, but also have 'downstream' effects upon later stages.

Other factors that make precision in research studies difficult include environmental influences on fertility and significant individual variation (for example, cycle length can vary from 21 to 38 days, between different women and for individuals). In addition, data obtained by recall, such as onset of menses, and timing of intercourse, is not always reliable.[19]

Nevertheless, in the face of these uncertainties and limitations, there is a significant body of research that has been conducted into the mechanisms of action of LNG.

HOW EFFECTIVE IS LNG AND HOW MIGHT IT WORK?

LNG's effectiveness at preventing clinical pregnancy is between 52% and 94% depending on the data used and the time of use relative to unprotected intercourse and ovulation.[20] The patient information for Levonelle One Step states that it "prevents about 84% of expected pregnancies when you take it within 72 hours of unprotected sex".[21]

Numerous studies show that the timing of LNG intake relative to ovulation is crucial for efficacy. When LNG is taken on the fertile days preceding ovulation, no clinical pregnancies result.[22,23,24] However, when taken on the day of ovulation or after, the number of pregnancies that would be expected to occur, do in fact occur.[25]

There are five primary ways by which LNG might exert its effect:

- Interference with sperm function, including interaction with the cervical mucus and ovum
- Inhibition or delay of ovulation (sufficient to ensure no viable sperm remain)
- Inhibition of embryo implantation by damaging endometrial receptivity
- Inhibition of embryo implantation by interference with its transport along the fallopian tube
- Inhibition of embryo implantation by interference with the corpus luteum and its role in preparing the endometrium

The first two of these are contraceptive and the latter three abortifacient.

DOES LNG AFFECT SPERM FUNCTION?

Studies on the effects of LNG on sperm have shown that there is no effect on sperm motility when the dose is a realistic equivalent of the dose normally found in the uterine environment after ingesting the standard EC dose (1.5mg).[26] Earlier studies did show an effect of LNG on sperm;[27] however, it is likely that the doses used were much higher than those found in the uterine environment. Indeed, it has been shown that the level of LNG in the uterus is much lower than that in the serum after 1.5mg.[28] In any case, given that LNG may be used up to 72 hours after intercourse, sperm may survive in the reproductive tract for all of this time and fertilise an ovum before any potential effect of LNG on sperm. In one study, sufficient quantities of viable sperm were found 24-48 hours after LNG intake.[29] The authors note that this also means there could have been no adverse effect on cervical mucus. Moreover, since sperm can reach the fallopian tube within 5 minutes,[30] any putative effect of LNG on cervical mucus would be too late.

LNG was also shown to have no effect on the acrosome reaction,[31,32] a process essential for fertilisation. Some of the confusion surrounding potential effects of LNG on sperm appears to be related to use of incorrect doses as well as to making unwarranted comparisons between LNG pills used for long term contraception and LNG for EC in a one-off dose. When both of these matters are considered, any effect on sperm function, if evident at all, cannot account for the efficacy of LNG.

Likewise, any effect of LNG on the interaction between sperm and ovum is highly unlikely. This had been proposed via elevated levels of the substance glycodelin A, which can inhibit fertilisation. However, research shows either such small changes in glycodelin A after LNG, far too low to inhibit fertilisation,[33] or no change at all.[34]

In summary, LNG does not appear to affect sperm or the process of fertilisation.

DOES LNG INHIBIT OR DELAY OVULATION?

If, as claimed, inhibition or delay of ovulation were the primary if not only mode of action of LNG, then there should be studies unequivocally proving this. But that is not the case. Instead, many show that LNG is not really that effective at inhibiting or delaying ovulation at all.

In the largest study by Noé and coworkers, 80% of women who took LNG prior to ovulation had nevertheless ovulated within 5 days.[35] These authors acknowledge that another mode of action must exist to account for the 100% efficacy of LNG at averting clinical pregnancy when taken in the late pre-ovulatory phase.

Numerous other studies have likewise found that LNG is not very effective at suppressing or delaying ovulation, especially in the late pre-ovulatory stage. Brache et al. found that LNG, taken when the dominant follicle was 18mm or greater (approximately day -2; ie 2 days before ovulation), was no more effective than placebo at suppressing or delaying ovulation.[36] The mean time from treatment to follicular rupture was 2.3 days for placebo, and 1.9 days for LNG.

Similar findings had already been reported by several other groups. For example, Hapangama *et al.* showed that 58% of women taking LNG in the pre-ovulatory stage still ovulated,[37] and Durand *et al.* showed that for women taking LNG 3 days prior to the LH surge (approximately day -4; ie 4 days prior to ovulation) all ovulated.[38,39] Croxatto *et al.* found that when LNG was taken at a follicular size of 12-14mm (approximately 4 days before expected ovulation; early pre-ovulatory), in 17% of cycles women still ovulated within 5 days. But this must be compared with the fact that even without LNG, ovulation only occurred in 44% of cycles. Hence, LNG's effectiveness was approximately 60%.[40]

A study by Okewole *et al.* found that LNG administered 3 days prior to ovulation delayed the LH surge and therefore ovulation; however, the delay may not have been sufficient to preclude fertilisation as 4 out of 8 subjects had delays of only between 1 and 4 days.[41] Women who took LNG one day before expected ovulation, ovulated normally.

Contrary to this body of research, two research groups found that LNG did effectively suppress ovulation. However, in one of these it *did* so when follicles were only 8mm in diameter, indicative of a very early stage many days prior to ovulation.[42] This would be consistent with the idea that LNG is more effective at suppressing ovulation the earlier it is taken relative to the usual time of ovulation. In the other study, Marions *et al.* found that ovulation was blocked in all subjects who took LNG 2 days prior to usual ovulation;[43] however, the study is somewhat unusual in that in a cycle prior to the LNG treatment cycle, the anti-progesterone agent mifepristone had been given to the same women. It would be unsurprising if use of a progesterone antagonist like mifepristone may have interfered with subsequent cycles.

To summarise the effects on ovulation, the majority of studies indicate that ovulation is delayed or suppressed somewhat when LNG is taken earlier in the cycle, perhaps at -4 days and earlier, but it becomes progressively less effective as the usual time of ovulation approaches. In fact, some studies show that LNG taken at day -2, or even -3, may be incapable of stopping ovulation at all, or delaying it enough to avoid fertilisation. It is likely that the majority of women who use LNG will be taking it on days -5 to 0 because of concerns they are in their fertile window. So, if the majority still ovulate and yet do not become pregnant (that is, clinically pregnant), something else besides inhibition or delay of ovulation must be happening.

DOES LNG AFFECT THE ENDOMETRIUM TO INHIBIT EMBRYO IMPLANTATION?

It is unlikely that LNG has a direct effect on the prepared endometrium, because when taken on the day of ovulation or after, it seems as if the expected number of clinical pregnancies result. Nevertheless, there have been several studies examining a possible effect. In a study using 3D endometrial constructs based upon endometrial biopsy taken at 4 to 5 days after the LH surge and therefore about 3-4 days after ovulation, no effect of LNG was found on a range of markers of receptivity.[44] In a separate study by the same authors, subjects ingested LNG on the day of ovulation or after, and then an endometrial biopsy was conducted several days later to analyse receptivity markers. There were significant changes in one of the key receptivity markers, the number of progesterone receptors, as well as in a substance called leukaemia inhibitory factor. While the authors acknowledge the central role of progesterone in regulating receptivity, they consider the changes not sufficient to affect receptivity to embryo attachment.[45]

Vargas and colleagues examined gene expression in biopsied endometrial tissue following exposure to LNG after ovulation. They found changes in the expression of only one gene among many and concluded that LNG does not significantly affect endometrial receptivity.[46]

In a different study designed to address the same question, endometrial tissue was biopsied 4 days after the LH surge (about 3 days post-ovulation), exposed to LNG and then assessed for attachment success with live human embryos over the next 5 days. For LNG exposed endometrium/embryos, 43% of embryos attached compared to 59% of controls. This suggest that LNG would affect implantation; however, the authors concluded that this was not a significant difference.[47]

In one of the studies referred to earlier, and in contrast to the studies just cited, LNG was taken by women prior to ovulation, and endometrial factors assessed.[48] However, rather than measuring a range of receptivity markers or expression of genes, this study only measured subsequent progesterone levels and the substance Glycodelin A. Glycodelin A, as noted earlier, can affect sperm-egg binding, but the changes in its levels caused by LNG are insufficient to have that effect. However, Glycodelin A also affects fetal-maternal interactions during the phase at which the embryo travels to the uterus and implants, and the finding that it is lowered during this phase by LNG (taken before ovulation), may indicate an adverse effect of LNG via Glycodelin A upon embryo survival. The authors note:

> … reduced endometrial glycodelin-A expression may indicate weakened immuno-suppressive microenvironment at the feto-maternal interface at the time of implantation.[49]

In other words, by reducing Glycodelin A, LNG may have interfered with the natural temporary immune suppression necessary to protect the embryo from attack as a foreign body. Such suppression is necessary to enable implantation.

In summary, although LNG does appear to have some direct effects upon the endometrium, they do not appear sufficient to impair embryo implantation and further development – at least when the endometrium has been able to naturally develop in readiness for implantation.

DOES LNG COMPROMISE EMBRYO TRANSPORT ALONG THE FALLOPIAN TUBE?

An embryo that has been conceived after the failure of LNG to stop ovulation must then travel along the fallopian tube at a rate sufficient to permit arrival at the endometrium during the fertile window. Delays could result in either embryo demise or possibly an ectopic pregnancy.

Several studies have shown that LNG slows fallopian tube motility by reducing what is termed the ciliary beat frequency (CBF) responsible for embryo movement.[50,51,52,53] One study went further and identified an effect of LNG upon the specific membrane channel known to regulate CBF.[54] Reduction in CBF could also provide an explanatory mechanism for embryo retention and possible increased risk of ectopic pregnancy; however, a 2010 review concluded no such risk existed.[55] Subsequent to that review, two studies have come to a different conclusion, that LNG *does* increase the risk of an ectopic pregnancy.[56,57]

In summary, while sufficient evidence to establish an increased risk of ectopic pregnancy after LNG is lacking, the evidence for reduced embryo movement, should ovulation and fertilisation proceed after LNG, is stronger. At this point in time there is no way of telling whether embryos conceived after LNG are inhibited from implantation because of reduced tubal motility, but it remains theoretically possible, and therefore a potential explanatory mechanism for an abortifacient effect.

CAN LNG INHIBIT ENDOMETRIAL PREPARATION AND LEAD TO EMBRYO DEMISE?

Because LNG is only modestly effective at inhibiting or delaying ovulation, and its effects on sperm or directly upon the endometrium are minimal, there must be another explanation as to why it is so effective at stopping clinical pregnancies when taken in the pre-ovulatory phase. Eggs will still be released, and sperm will often still be present; hence fertilisation can occur, and embryos will likely be conceived. Why do they not continue to clinical pregnancy?

One possible explanation is that the pre-ovulatory use of LNG impairs the processes that prepare the endometrium for embryo implantation. This impairment has been termed 'corpus luteum dysfunction'.[58] The corpus luteum is the remnant of the ovarian follicle after the egg had been released, and it plays a central role in producing progesterone and estrogen for the proper maintenance of an environment optimal for embryo transport, embryo/maternal interactions, implantation, and continued development.[59] Many studies have identified disruption by pre-ovulatory LNG of the luteal (ie post-ovulatory) phase, which is indicative of an impaired corpus luteum. [60,61,62,63,64,65,66,67] LNG blunts the LH surge, which then leads to lowered progesterone. While the exact mechanism of action is not clear, what is clear is that the LH surge is reduced in magnitude and duration. The subsequent drop in progesterone level has also been measured, and since progesterone is known to prepare the endometrium for implantation, an embryo might arrive and be unable to properly attach to the uterine lining, leading to its demise. The other indicator of compromised luteal function is that the post-ovulatory phase is shortened by LNG, a finding which may be related to the increased vaginal bleeding it produces. Such an increase indicates premature endometrial sloughing, an action that could also inhibit implantation.[68] Endometrial sloughing typically occurs at a point in the cycle when no embryo has implanted, but its premature occurrence effectively strips the uterine lining of its ability to host an embryo.

CONCLUSION

In summary, a complete description of the mechanisms of action of LNG has yet to be determined, but the evidence that does exist suggests it has both pre- and post- fertilisation effects. In other words, it is most likely contraceptive *and* abortifacient. What might occur in any individual case depends on the time of intercourse relative to ovulation, and the time of LNG ingestion relative to both. Which of the two actions may have been responsible for the avoidance of a clinical pregnancy cannot therefore be known with certainty.

More studies clearly need to be done to ascertain how LNG works, but perhaps the most useful addition to the field would be a test that shows whether conception has occurred, perhaps accurate to within 48 hours of fertilisation. That would then indicate the presence of an embryo and whether continued development is able to occur if LNG is taken in the lead up to the expected time of ovulation. Significant research has already identified the release of a substance called *early pregnancy factor* that could serve such a purpose,[69] but it would take significant investment in time and resources to utilise it in a research study. Perhaps just as important is the will to undertake such a study, given the potential ramifications.

One thing is certain, the various authorities responsible for providing women with the best available evidence about LNG should stop being so definitively reassuring that it is only contraceptive. Unfortunately, continuing to do so only serves to undermine the trust that is so central to their role.

ENDNOTES

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8. In their decisional memorandum explaining the labelling changes, the FDA states, "Mechanism of action information is not needed for the safe or effective use of nonprescription drugs in general, and not for levonorgestrel emergency contraception in particular." It is not clear why LNG should be a special case, but nevertheless, contrary to their usual practice for other non-prescription drugs, the FDA are prepared to provide this information for LNG. It would be useful if there was an explanation as to why. See https://www.accessdata.fda.gov/drugsatfda_docs/label/2022/021998Orig1s005SumR.pdf, p 9, Accessed 7 Jan 2026.
9. Foundation Consumer Healthcare (December 23, 2022) Press Release. Available from: https://foundationch.com/press-release/2022-12-23/ Accessed 5 Jan 2026.
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23. Noé G *et al.* (2010) Contraceptive efficacy of emergency contraception with levonorgestrel given before or after ovulation. *Contraception* 81:414-420.
24. Noé G *et al.* (2011) Contraceptive efficacy of emergency contraception with levonorgestrel given before or after ovulation. *Contraception* 84:486-492.

25. There is an important qualifier to studies that estimate clinical pregnancy rates, many based on the calculations by Wilcox and coworkers [Wilcox AJ *et al.* (1995) Timing of sexual intercourse in relation to ovulation. Effects on the probability of conception, survival of the pregnancy, and sex of the baby. *New Engl J Med* 333(23):1517-1521; Wilcox AJ *et al.* (1998) Post-ovulatory ageing of the human oocyte and embryo failure. *Human Reproduction* 13(2):394-397]. In their 1995 paper, Wilcox *et al.* derived expected *conception* rates that climbed to around 35% for days -2, -1 and 0 (the day of ovulation). However, in their 1998 paper, they accounted for early embryo loss as well as oocyte aging on the day of ovulation to derive figures for *clinical pregnancies* of 29%, 27% and 8% for days -2, -1 and 0, respectively. The authors acknowledge that "the confidence intervals around all these estimates are very wide". Hence, using data from Wilcox's studies is not without its limitations.

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39. In December 2022, the FDA produced a decisional memorandum to explain why they agreed to changes to the consumer information leaflet. It now explicitly states that LNG does not have a post fertilisation, that is abortifacient effect. In that memorandum they refer to the paper by Durand *et al.*, correctly describing how the researchers found no effect of LNG on ovulation in a cohort of women who received LNG several days before the expected day of ovulation. But confoundingly, they then summarise Durand's work by stating, "Overall, the data reported by Durand supported prevention of ovulation by LNG." See https://www.accessdata.fda.gov/drugsatfda_docs/label/2022/021998Orig1s005SumR.pdf, p 9, Accessed 7 Jan 2026.

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