Sperm drastically change their flagellar movement in response to the surrounding physical and chemical environment. Testicular sperm are immotile; however, they gain the competence to initiate motility during passage through the male reproductive tract. Once ejaculated, the sperm are activated and promptly initiate motility. Unlike mammals, ejaculated sperm in birds are stored in specialized tubular invaginations referred to as sperm storage tubules (SSTs), located between the vagina and uterus, before fertilization. The resident sperm in the SSTs are in a quiescent state and then re-activated after release from the SSTs. It is thought that avian sperm can undergo motility change from quiescent to active state twice; however, the molecular mechanism underlying sperm motility regulation is poorly understood. In this short review, we summarize the current understanding of sperm motility regulation in male and female bird reproductive tracts. We also describe signal transduction, which regulates sperm motility, mainly derived from in vitro studies.

Key words: avian sperm, sperm motility, sperm storage

Introduction

In vertebrates, sperm are produced as self-mobile tiny cells that carry male haploid DNA for successful fertilization. After production in testes, sperm initiate a journey to reach ovulated oocytes, which are localized deep inside the female reproductive tract. Before fertilization, sperm descend the male genital tract and sperm maturation proceeds. Then, they are ejaculated together with seminal plasma and transferred into the female genital tract by copulation. After copulation, sperm then ascend higher into the female genital tract and finally reach the oocyte fertilization site. Although as many as one hundred million sperm are ejaculated into the female reproductive tract during each copulation, only a small fraction that arrives at the site of fertilization is capable of fusing with ovulated ova (reviewed by Matsuzaki and Sasanami, 2017). From this perspective, the fertilization process can be considered highly competitive for sperm compared to oocytes. To compete with rival sperm, sperm motility is one of the most important traits because highly motile sperm can swim faster than less motile sperm (Rosengrave et al., 2008; Kim et al., 2017). It is known that sperm motility is generated by axonemal motor protein dynein, which hydrolyzes ATP to drive microtubule sliding (Gibbons, 1988; Inaba, 2003).

In internal fertilizers, successful fertilization also depends on the timely arrival of both gametes at the site of fertilization; however, de-coupling of ovulation timing and insemination is common in the majority of species. Females of such species can store sperm in reproductive tracts until eggs are ready to be fertilized. This phenomenon is common in many non-mammalian animals, including insects, fish, amphibians, reptiles, and birds (Birkhead and Møller, 1993; Holt, 2011). In birds, sperm storage tubules (SSTs), which are simple tubular invaginations located between the vagina and uterus, serve as sperm storage sites (Bakst et al., 1994; Sasanami et al., 2013; Matsuzaki and Sasanami, 2017). Once ejaculated, sperm migrate to and are subsequently stored in the lumen of SSTs, where they can remain without loss of fertilization capacity for long periods of time (up to 15 weeks) at normal body temperatures (41°C) (Birkhead and Møller, 1992; Bakst, 2011). Of particular interest in sperm motility regulation is that avian sperm can undergo motility alteration from quiescent to active state twice. First, sperm...
motility initiation occurs after ejaculation and is then inactivated while sperm reside in the SSTs. Motility is then thought to be re-activated after release from the SSTs. Therefore, a molecular switch, which controls motility activation and inactivation in response to extracellular stimuli, may operate in avian sperm; however, the underlying mechanism is yet to be elucidated.

In this review, we summarize the current understanding of sperm motility regulation in avian reproductive tracts (Fig. 1). We also describe signal transduction pathways that regulate sperm motility, mainly reported in vitro studies.

**Sperm Motility Regulation in the Male Reproductive Tract**

In natural mating or artificial insemination, sperm motility is integral for the success of fertilization. Howarth (1983) reported that testicular sperm showed limited motility, and no fertilized egg was obtained when they were inseminated into vaginas (Howarth, 1983). On the other hand, epididymal sperm showed medium motility (49% of sperm were motile) and those obtained from the vas deferens exhibited equivalent motility (88%) to ejaculated sperm when suspended in a sperm extender. Considering this, sperm descending the male reproductive tract gradually acquire motility competence in the epididymis and fully develop this trait after reaching the vas deferens. However, the factors/mechanisms that lead to sperm motility competence in the male reproductive tract are not understood. Few studies are available, and our current understanding is extremely limited. Ashizawa and his colleague tested the effects of seminal plasma on the motility of washed sperm in chickens. They demonstrated that the addition of different doses of seminal plasma enhanced sperm motility in vitro (Ashizawa and Wishart, 1987). Sperm motility reduction in the absence of seminal plasma is not thought to be due to a reduction in energy metabolism because ATP contents in the cells showed similar levels irrespective of the presence or absence of seminal plasma (Ashizawa and Wishart, 1987). These results indicate the presence of factors in seminal plasma that regulate sperm motility. Although it is not known whether these substances alter sperm motility competence or simply enhance sperm motility itself, fractionation studies on chicken seminal plasma indicated that the factor affecting sperm motility was a low molecular weight substance (Ashizawa and Okauchi, 1984; Ashizawa and Wishart, 1987). Unfortunately, the nature of this substance is un-
known.

As mentioned above, sperm stored in vas deferens have motility competence; however, the motility initiation is arrested. We also confirmed that vas deferens sperm in quails are immotile inside; however, they quickly gain motility when suspended in sperm extender (Matsuzaki et al., 2017). Currently, it is unclear how sperm motility is arrested in the vas deferens. In internal fertilizers such as marine invertebrates and fish, sperm are immotile before spawning, but they are immediately activated by specific chemicals or environmental cues, such as osmolality changes (Inaba, 2003; Yoshida et al., 2008). In ascidians, a sulfated steroid, referred to as sperm activating and attracting factor (SAAF), is released from the eggs and the sperm sense the SAAF via plasma membrane Ca\(^{2+}\)-ATPase to initiate motility (Yoshida et al., 2002; Nomura et al., 2004). In freshwater teleosts, sperm motility is initiated when sperm are diluted in a hypotonic solution, whereas a hypertonic solution effectively activates sperm motility in marine teleosts (Morisawa and Suzuki, 1980). In the newt Cynops pyrrhogaster, a protein localized in the outer layer of the egg jelly, referred to as a sperm motility-initiating substance, was reported to initiate sperm motility (Watanabe et al., 2011). Our preliminary experiments in Japanese quail showed that the ATP contents in vas deferens sperm were very low, but immediately (within 30 seconds) increased as sperm motility was initiated (data not shown). These results indicate the possibility that ATP production in vas deferens sperm is inhibited by an unknown mechanism and that such inhibitory effects are not observed when the sperm are released outside the body. However, further studies are required to elucidate the initiation system in avian sperm motility.

**Sperm Motility Regulation in Female Reproductive Tract**

During copulation, males release fully mature sperm into the female vagina. In birds, spermatozoa initially reside in oviductal sperm storage sites, SSTs, before encountering an ovulated ovum. Sperm are stored in the SSTs located in the uterovaginal junction (UVJ) and released according to the timing of ovulation. Before entering SSTs, ejaculated sperm are selected in the vagina, most likely via sperm ejection through female defecation. In turkeys, more than 80% of the sperm are ejected from the vagina soon after mating (Howarth, 1983). In addition, less than 1% of sperm inseminated into the vagina enter the SST (Bakst et al., 1994). Sperm artificially introduced into the vagina of a chicken reached the SST within an hour (Das et al., 2009), and the intrinsic motility of sperm may be an important factor in the uptake into the SST (Froman, 2003). In fact, it was reported that sperm with poor motility showed less ability to fill the SSTs, and the paternity of embryos was biased towards males with high sperm motility when a sperm mix with similar sperm numbers from two males was artificially inseminated into females (unpublished data). Although there is no doubt that sperm motility is essential for better fertilization success, we have no information as to whether sperm motility is regulated by factors derived from the female reproductive tract.

We, and others, observed that sperm residing in SSTs are quiescent in motility (Bakst, 1987; Matsuzaki et al., 2015), and this phenomenon is a long-standing enigma in the field of avian reproduction. We recently demonstrated that the luminal environment of SSTs is acidic due to the presence of large amounts of lactic acid (approximately 13 mM) released from the SSTs under hypoxic conditions (Matsuzaki et al., 2015). This acidic condition inhibits the ATPase activity of dynein, the motor protein of sperm flagellum (Matsuzaki et al., 2015). In this condition, the pH of sperm cytosol also declines to around 5.4, coinciding with the extracellular pH. The hypoxic condition created by the SSTs also decreases sperm mitochondrial activity (Matsuzaki et al., 2015). Thus, sperm motility in the SSTs is highly arrested due to less energy production and consumption. This system appears reasonable regarding sperm storage for longer periods of time because reduced sperm respiration in the SSTs leads to less production of reactive oxygen species and thus enables minimal sperm damage. In contrast to our experimental evidence regarding arrested sperm motility in the SSTs, Froman (2003) suggested another model in which sperm maintained their motility inside of SSTs in chickens (Froman, 2003). He suggested that sperm sustain their location against the reverse flow (from the base of the SSTs to orifice) generated by SSTs, and are egressed from the SSTs when the sperm swimming velocity falls below the flow. Furthermore, Ahammad et al. (2011) reported that chicken sperm gain the ability to bind with SST epithelial cells during the passage though the male reproductive tract, and this binding plays a role in sperm storage (Ahammad et al., 2011). Although the speculation of such a ligand-receptor interaction model is interesting to investigate, we, and others, observed that sperm residing in SSTs form a bundle (Bakst, 1983; Sasanami et al., 2013), and the cells located in the center of the bundle are unable to contact the SST epithelium. We have no explanation for this inconsistency between chicken and quail sperm.

The sperm stored in the SSTs are released according to the time of ovulation and reach the fertilization site. Females have been shown to actively control the timing of sperm release. When females with sperm-filled SSTs were intravenously injected with progesterone, most of the sperm were released within 1 h post-injection (Ito et al., 2011). The circulating progesterone in poultry, including Japanese quail, reaches a peak around 4 to 6 h before the next ovulation (Marshay et al., 1976; Doi et al., 1980; Etches and Cheng, 1981). Therefore, it is reasonable to suppose that the two events, sperm release from the SST and ovulation, are stimulated simultaneously by progesterone to achieve efficient fertilization. Furthermore, we found that the surface epithelial cells of UVJ express heat shock protein 70 (HSP70), and we hypothesize that this protein stimulates sperm motility to facilitate sperm migration in the oviduct after its release from SSTs (Hiyama et al., 2014). Additionally, we found that bacterially expressed HSP70 activates...
flagellar movement in sperm and that recombinant HSP70 binds to the surface of sperm by interacting with voltage-dependent anion channel protein 2 (VDAC2) (Hiyama et al., 2014). Furthermore, injection of anti-HSP70 antibody into the quiescent state at 40°C temperature. Alternatively, the alkalization of sperm pH approximately 0.3 units lower than pHi (pHi) ranging from 7.3–10.1, whereas sperm were in a quiescent state at 40°C in a medium with pHe below 8.1 (Ashizawa et al., 1994c). Because pHi at 40°C is approximately 0.3 units lower than pHi at 30°C, an acidic pHi at 40°C may play a role in motility immobilization at body temperature. Alternatively, the alkalization of sperm pHi is important for sperm motility activation in chickens. To further understand this unique phenomenon in chicken sperm, the authors investigated flagellar motility in demembranated sperm in which sperm were treated with the non-ionic detergent, TritonX-100. They found that the de-membranated sperm expressed similar motility patterns to intact sperm regarding reversible immobilization of motility (Ashizawa et al., 1989b), indicating that de-membranated spermatozoa are a suitable model for investigating the direct effects of various inhibitors/activators that are not able to pass through the plasma membrane by sperm flagellar movements in vitro. Using this system, it was found that complex signal transduction machinery exists in sperm motility regulation in chickens. For instance, the addition of recombinant protein phosphatase type 1 (PP-1), the specific activator of protein kinase C (PKC), or the substrate peptides for mitogen-activated protein kinase (MAPK) or p34cdc2 kinase markedly decreased the motility of de-membranated spermatozoa at 30°C, indicating that the protein phosphorylation/de-phosphorylation may be involved in sperm motility regulation (Ashizawa et al., 1994d, 1997a, 2006). A marked difference in the phosphorylation status of 116-, 86-, 79-, 50-, and 29-kDa proteins was observed after the addition of these inhibitors (Ashizawa et al., 1995, 1997a, b). Recently, we also studied the effects of various inhibitors of protein kinases in quail spermatozoa (Matsuzaki et al., 2017). We employed four protein kinase inhibitors: bisindolylmaleimide II (BisII), a potent competitive protein kinase C (PKC) inhibitor (Mahata et al., 2002); bisindolylmaleimide V (BisV), a weak inhibitor of PKC (Mahata et al., 2002); H-89, a potent cell-permeable inhibitor of protein

Mechanism Regulating Sperm Motility

It is known that the flagellar movement of sperm is driven by the sliding of microtubules with motor protein dynein ATPase (Gibbons, 1988, 1996; Inaba, 2007). However, much is unresolved regarding the regulatory mechanism of sliding events in the axoneme. Ashizawa and colleagues observed a reversible immobilization of chicken sperm motility in which sperm suspended in a simple salt solution without Ca2+ were immotile at body temperature (40°C), but motility was instantly restored when the incubation temperature decreased to 30°C (Munro, 1938; Ashizawa and Nishiyama, 1978). Another group reported that this reversible activation/inactivation of motility is also observed in drake sperm and partially in turkey sperm, but not in quail sperm (Wishart and Wilson, 1999). In chicken sperm, motility inhibition at 40°C was quickly recovered when Ca2+ was included in the incubation mixture (Ashizawa et al., 1989a). The depletion of Ca2+ from the medium by adding Ca2+ chelating reagents disturbed sperm motility even at 30°C. Therefore, it is considered that Ca2+ is essential for the maintenance of chicken sperm motility, as also reported in the sperm of many animals (Ashizawa et al., 1994b). Later, it was found that motility inactivation at body temperature was also canceled by an increase in intracellular pH (pHi) of the sperm (Ashizawa et al., 1994c). At 30°C, sperm motility was activated in a medium with various extracellular pH (pHe) ranging from 7.3–10.1, whereas sperm were in a quiescent state at 40°C in a medium with pHe below 8.1 (Ashizawa et al., 1994c). Because pHe at 40°C is approximately 0.3 units lower than pHi at 30°C, an acidic pHe at 40°C may play a role in motility immobilization at body temperature. Alternatively, the alkalization of sperm pHe is important for sperm motility activation in chickens. To further understand this unique phenomenon in chicken sperm, the authors investigated flagellar motility in demembranated sperm in which sperm were treated with the non-ionic detergent, TritonX-100. They found that the de-membranated sperm expressed similar motility patterns to intact

Fig. 2. Light micrograph of the avian sperm head, mid-piece, and tail. (A) Japanese quail sperm. The junction of the head and midpiece or midpiece and tail are clearly differentiated. (B) Chicken sperm. The junction of the head and midpiece is unclear. Bar=50μm.
the flagellar movement of fowl spermatozoa (Ashizawa et al., 1994). The corresponding author would like to express her sincere gratitude to all her collaborators and students, as well as her family. This work was supported, in part, by a Grant-in-Aid for Young Scientist (20K15648 to MM), a Grant-in-Aid for Scientific Research (B) (General) (17H03902 to TS), and a Grant-in-Aid for Challenging Exploratory Research (20K21368 to TS).

Conflicts of Interest

The authors declare no conflict of interest.

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Ahammad MU, Nishino C, Tatemoto H, Okura N, Kawamoto Y, Okamoto S and Nakada T. Maturational changes in the spermic fertilization and oviductal sperm storage, are employed for successful fertilization in avian species. Several important phenomena and molecules that regulate avian fertilization have been discovered, but our understanding of the precise mechanism has not advanced significantly because there are no efficient methods to produce gene-manipulated birds. With the use of modern technology, such as the CRISPR/Cas9 system, we expect the production of transgenic and gene-knockout birds in the near future. It is possible that reverse genetics will advance our understanding of the mechanisms of avian fertilization, including sperm motility regulation.

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Conclusion

In this short review, we summarized our current understanding regarding sperm motility regulation in birds. Avian fertilization systems are quite different from those of mammalian species because unique systems, such as poly-

Table 1. Effects of various chemicals on chicken and quail sperm motility in the presence or absence of Ca$^{2+}$ in vitro at 40°C

| Chemicals | Effects |
|-----------|---------|
| EGTA      | Ca$^{2+}$ chelator |
| BAPTA-AM  | Intracellular Ca$^{2+}$ chelator |
| PD 150606 | Ca$^{2+}$-dependent calpain inhibitor |
| Y-27632   | Ca$^{2+}$-dependent Rho-kinase inhibitor |
| W-7       | Calmodulin antagonist |
| W-5       | Calmodulin antagonist |
| Trifluoperazine | Calmodulin antagonist |
| Calyculin A | PP1 and PP2A inhibitor |
| Okadaic acid | PP1 and PP2A inhibitor |
| LY294002  | PI3-kinase inhibitor |
| 1L-6-Hydroxymethyl-chiro-inositol 2-(R)-2-O-methyl-3-O-octadecylicarbonate | Akt inhibitor |
| H89       | PKA inhibitor |
| SC-9      | PKC activator |
| OAG       | PKC activator |
| H-7       | PKC inhibitor |
| Bis II    | PKC inhibitor |

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